

Trying 3106016892...Open

Welcome to STN International! Enter x:x

LOGINID:sssptal617srh

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * Welcome to STN International * * * * *

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America
NEWS 2 Dec 17 The CA Lexicon available in the CAPLUS and CA files
NEWS 3 Feb 06 Engineering Information Encompass files have new names
NEWS 4 Feb 16 TOXLINE no longer being updated
NEWS 5 Apr 23 Search Derwent WPINDEX by chemical structure
NEWS 6 Apr 23 PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS 7 May 07 DGENE Reload

NEWS EXPRESS April 18 CURRENT WINDOWS VERSION IS V6.0,
CURRENT MACINTOSH VERSION IS V5.0C (ENG) AND V5.0JB (JP),
AND CURRENT DISCOVER FILE IS DATED 04/06

NEWS HOURS STN Operating Hours Plus Help Desk Availability

NEWS INTER General Internet Information

NEWS LOGIN Welcome Banner and News Items

NEWS PHONE Direct Dial and Telecommunication Network Access to STN

NEWS WWW CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 11:31:54 ON 14 MAY 2001

=> fil reg

COST IN U.S. DOLLARS

SINCE FILE

ENTRY

TOTAL

SESSION

FULL ESTIMATED COST

0.45

0.45

FILE 'REGISTRY' ENTERED AT 11:33:36 ON 14 MAY 2001

USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.

PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

COPYRIGHT (C) 2001 American Chemical Society (ACS)

STRUCTURE FILE UPDATES: 13 MAY 2001 HIGHEST RN 335276-85-2

DICTIONARY FILE UPDATES: 13 MAY 2001 HIGHEST RN 335276-85-2

TSCA INFORMATION NOW CURRENT THROUGH January 11, 2001

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Structure search limits have been increased. See HELP SLIMIT for details.

=> s misoprostol/cn
L1 1 MISOPROSTOL/CN

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	4.11	4.56

FILE 'STNGUIDE' ENTERED AT 11:34:08 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.00	4.56

FILE 'HCAPLUS' ENTERED AT 11:35:06 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

Copyright of the articles to which records in this database refer is held by the publishers listed in the PUBLISHER (PB) field (available for records published or updated in Chemical Abstracts after December 26, 1996), unless otherwise indicated in the original publications.

FILE COVERS 1947 - 14 May 2001 VOL 134 ISS 21
FILE LAST UPDATED: 13 May 2001 (20010513/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

This file supports REGISTRY for direct browsing and searching of all substance data from the REGISTRY file. Enter HELP FIRST for more information.

HCAplus now provides online access to patents and literature covered in CA from 1947 to the present. On April 22, 2001, bibliographic information and abstracts were added for over 2.2 million references published in CA from 1947 to 1966.

=> s misoprostol or l1
845 MISOPROSTOL
727 L1
L2 882 MISOPROSTOL OR L1

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (a) (hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or respon? or anhedonia)

UNMATCHED LEFT PARENTHESIS 'S) ((SEX?'

The number of right parentheses in a query must be equal to the number of left parentheses.

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (a) (hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or respon? or anhedonia))

103473 FEMALE

47254 FEMALES

130450 FEMALE

(FEMALE OR FEMALES)

4603 WOMAN

9 WOMANS

58044 WOMEN

23 WOMENS

60685 WOMAN

(WOMAN OR WOMANS OR WOMEN OR WOMENS)

58044 WOMEN

23 WOMENS

58053 WOMEN

(WOMEN OR WOMENS)

1029 GIRL

2705 GIRLS

3554 GIRL

(GIRL OR GIRLS)

328 LADY

132 LADIES

457 LADY

(LADY OR LADIES)

108098 SEX?

5157 GENITAL

154 GENITALS

5267 GENITAL

(GENITAL OR GENITALS)

792 HYPOACTIV?

123498 DESIR?

138810 SATISFACT?

51 ORGASM

3156 AROUSAL?

2173 SENSATION?

1553151 RESPON?

121 ANHEDONIA

L3 145 (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR GENITAL

AROUSAL?

) (A) (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR

OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s scan

19854 SCAN

9143 SCANS

L4 27636 SCAN

(SCAN OR SCANS)

=> d scan

L4 27636 ANSWERS HCAPLUS COPYRIGHT 2001 ACS

IC ICM H01L021-66

ICS H01L021-66; G01B015-00; G01B015-04; G01N001-28; G01N001-32;

G01N023-04; G01N023-225; H01J037-22; H01J037-26; H01L021-3065

TI Defective inspection + defective method for analyzing of
semiconductor device pattern, survey instrument of defective inspection +
failure analysis system, and semiconductor device pattern of
semiconductor
device pattern. [Machine Translation].

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):2

L4 27636 ANSWERS HCAPLUS COPYRIGHT 2001 ACS
IC ICM G02F001-1343
ICS G02F001-1337; G09F009-30; G09F009-35
TI Liquid crystal display. [Machine Translation].

L4 27636 ANSWERS HCAPLUS COPYRIGHT 2001 ACS
IC ICM G01R031-02
ICS H05K003-00
TI Etched circuit substrate inspection vessel. [Machine Translation].

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):1

L4 27636 ANSWERS HCAPLUS COPYRIGHT 2001 ACS
IC ICM G01M011-00
ICS G02F001-13
TI Grade inspection method and its grade survey instrument of plane surface
display. [Machine Translation].

HOW MANY MORE ANSWERS DO YOU WISH TO SCAN? (1):0

=> s ((sex? or genital) (a) (hypoactiv? or desir? or satisfact? or orgasm or
arousal? or sensation? or respon? or anhedonia))

108098 SEX?
5157 GENITAL
154 GENITALS
5267 GENITAL
(GENITAL OR GENITALS)
792 HYPOACTIV?
123498 DESIR?
138810 SATISFACT?
51 ORGASM
3156 AROUSAL?
2173 SENSATION?
1553151 RESPON?
121 ANHEDONIA
L5 595 ((SEX? OR GENITAL) (A) (HYPOACTIV? OR DESIR? OR SATISFACT? OR
ORGASM OR AROUSAL? OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s ((sex? or genital) (s) (hypoactiv? or desir? or satisfact? or orgasm or
arousal? or sensation? or respon? or anhedonia))

108098 SEX?
5157 GENITAL
154 GENITALS
5267 GENITAL
(GENITAL OR GENITALS)
792 HYPOACTIV?
123498 DESIR?
138810 SATISFACT?
51 ORGASM
3156 AROUSAL?
2173 SENSATION?
1553151 RESPON?

121 ANHEDONIA
L6 12767 ((SEX? OR GENITAL) (S) (HYPOACTIV? OR DESIR? OR SATISFACT? OR
ORGASM OR AROUSAL? OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia)

UNMATCHED LEFT PARENTHESIS 'S) ((SEX?'

The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia))

103473 FEMALE

47254 FEMALES

130450 FEMALE

(FEMALE OR FEMALES)

4603 WOMAN

9 WOMANS

58044 WOMEN

23 WOMENS

60685 WOMAN

(WOMAN OR WOMANS OR WOMEN OR WOMENS)

58044 WOMEN

23 WOMENS

58053 WOMEN

(WOMEN OR WOMENS)

1029 GIRL

2705 GIRLS

3554 GIRL

(GIRL OR GIRLS)

328 LADY

132 LADIES

457 LADY

(LADY OR LADIES)

108098 SEX?

5157 GENITAL

154 GENITALS

5267 GENITAL

(GENITAL OR GENITALS)

792 HYPOACTIV?

123498 DESIR?

138810 SATISFACT?

51 ORGASM

3156 AROUSAL?

2173 SENSATION?

1553151 RESPON?

121 ANHEDONIA

L7 1933 (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENITAL

) (S) (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR
AROUSAL?
OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s 12 and 17

L8 2 L2 AND L7

=> d ti tot

L8 ANSWER 1 OF 2 HCAPLUS COPYRIGHT 2001 ACS
TI Use of **misoprostol** or/and **misoprostol** acid for
preparing drug in order to cure sexual dysfunction in women

L8 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2001 ACS
TI Methods, compositions, and kits for enhancing **female
sexual desire** and **responsiveness** using
prostaglandins

=> d ibib abs 2

L8 ANSWER 2 OF 2 HCAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:282087 HCAPLUS
DOCUMENT NUMBER: 130:321230
TITLE: Methods, compositions, and kits for enhancing
female sexual desire and
responsiveness using prostaglandins
INVENTOR(S): Neal, Gary W.
PATENT ASSIGNEE(S): Androsolutions, Inc., USA
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9920266	A1	19990429	WO 1998-US21631	19981020
W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
AU 9896952	A1	19990510	AU 1998-96952	19981020
EP 1028720	A1	20000823	EP 1998-951063	19981020
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI			

PRIORITY APPLN. INFO.: US 1997-954122 A 19971020
WO 1998-US21631 W 19981020

AB Topical application of a prostaglandin directly to the clitoris is effective for enhancing **female sexual desire** and **responsiveness**. Kits and pharmaceutical compns. contg. the prostaglandins are claimed as well. The pharmaceutical compns. may contain at least one coagent as well selected from the group consisting of 15-hydroxyprostaglandin dehydrogenase inhibitors, ACE inhibitors, nitro vasodilators, alpha blockers, yohimbine, labetalol, carvedilol, bucindolol, phosphodiesterase inhibitors, muscarinic agents, dopaminergic agonists, ergot alkaloids, opiate antagonists, and polypeptide neurotransmitters.

REFERENCE COUNT: 2
REFERENCE(S): (1) Hadley; US 5576290 A 1996 HCAPLUS
(2) Roy, A; British Journal of Urology 1989, V64,

P180

MEDLINE

=> fil stng

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
26.66	31.22

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
-0.59	-0.59

CA SUBSCRIBER PRICE

FILE 'STNGUIDE' ENTERED AT 11:42:34 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: May 11, 2001 (20010511/UP).

=> file medline caplus embase biosis uspatfull

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
0.00	31.22

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
0.00	-0.59

CA SUBSCRIBER PRICE

FILE 'MEDLINE' ENTERED AT 11:52:55 ON 14 MAY 2001

FILE 'CAPLUS' ENTERED AT 11:52:55 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'EMBASE' ENTERED AT 11:52:55 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 11:52:55 ON 14 MAY 2001
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'USPATFULL' ENTERED AT 11:52:55 ON 14 MAY 2001
CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

=> e neal, gary/au

E1	9	NEAL ZANE C/AU
E2	1	NEAL ZANE CLAY/AU
E3	0 -->	NEAL, GARY/AU
E4	1	NEALAND S/AU
E5	18	NEALE A/AU
E6	4	NEALE A B/AU
E7	8	NEALE A C/AU
E8	18	NEALE A D/AU
E9	1	NEALE A E T/AU
E10	13	NEALE A J/AU
E11	1	NEALE A M/AU
E12	4	NEALE A R/AU

=> e neal gary/au

E13 2 NEAL GARDNER S/AU
 E14 2 NEAL GARRISON R/AU
 E15 0 --> NEAL GARY/AU
 E16 1 NEAL GARY F/AU
 E17 1 NEAL GARY R/AU
 E18 10 NEAL GARY W/AU
 E19 3 NEAL GENEVIEVE S/AU
 E20 1 NEAL GEORGE/AU
 E21 2 NEAL GEORGE F/AU
 E22 1 NEAL GEORGE H/AU
 E23 1 NEAL GERALD V/AU
 E24 2 NEAL GERALD WAYNE/AU

=> e e18

E25 1 NEAL GARY F/AU
 E26 1 NEAL GARY R/AU
 E27 10 --> NEAL GARY W/AU
 E28 3 NEAL GENEVIEVE S/AU
 E29 1 NEAL GEORGE/AU
 E30 2 NEAL GEORGE F/AU
 E31 1 NEAL GEORGE H/AU
 E32 1 NEAL GERALD V/AU
 E33 2 NEAL GERALD WAYNE/AU
 E34 1 NEAL GERTRUDE M/AU
 E35 3 NEAL GORDON/AU
 E36 62 NEAL GORDON E/AU

=> s e18

L9 10 "NEAL GARY W"/AU

=> d ti so tot

L9 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2001 ACS
 TI Methods and compositions for preventing and treating urinary tract disorders
 SO PCT Int. Appl., 32 pp.
 CODEN: PIXXD2

L9 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2001 ACS
 TI Prostaglandin derivatives for preventing and treating prostate disorders
 SO PCT Int. Appl., 40 pp.
 CODEN: PIXXD2

L9 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2001 ACS
 TI Methods, compositions, and kits for enhancing female sexual desire and responsiveness using prostaglandins
 SO PCT Int. Appl., 51 pp.
 CODEN: PIXXD2

L9 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2001 ACS
 TI male erectile dysfunction with a prostaglandin vasodilator and a 15-hydroxyprostaglandin dehydrogenase inhibitor, and suppository composition
 SO PCT Int. Appl., 67 pp.
 CODEN: PIXXD2

L9 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2001 ACS
 TI Insulin degradation by human skeletal muscle
 SO Biochim. Biophys. Acta (1982), 719(2), 259-66
 CODEN: BBACAQ; ISSN: 0006-3002

L9 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2001 ACS
TI Catecholborane (1,3,2-benzodioxaborole). A versatile reducing agent
SO J. Org. Chem. (1977), 42(3), 512-17
CODEN: JOCEAH

L9 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2001 ACS
TI Organoboranes as alkylating agents
SO J. Chem. Educ. (1976), 53(9), 549-50
CODEN: JCEDA8

L9 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2001 BIOSIS
TI Methods for treating male erectile dysfunction.
SO Official Gazette of the United States Patent and Trademark Office
Patents,
(Aug. 15, 2000) Vol. 1237, No. 3, pp. No Pagination. e-file.
ISSN: 0098-1133.

L9 ANSWER 9 OF 10 USPATFULL
TI Methods for treating male erectile dysfunction

L9 ANSWER 10 OF 10 USPATFULL
TI Method and means for linearizing a voltage controlled oscillator sweep
generator

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia))

<-----User Break----->

u

SEARCH ENDED BY USER
SEARCH ENDED BY USER

=> fil medline embase biosis uspatfull		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	13.43	44.65
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'MEDLINE' ENTERED AT 11:57:31 ON 14 MAY 2001

FILE 'EMBASE' ENTERED AT 11:57:31 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 11:57:31 ON 14 MAY 2001
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'USPATFULL' ENTERED AT 11:57:31 ON 14 MAY 2001
CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia))

3 FILES SEARCHED...

L10 43446 (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENITAL

AROUSAL?) (S) (HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR
OR SENSATION? OR RESPON? OR ANHEDONIA))

=> s 11 or misoprostol
L11 6378 L1 OR MISOPROSTOL

=> s 110 and 111
L12 1 L10 AND L11

=> d ibib abs

L12 ANSWER 1 OF 1 USPATFULL

ACCESSION NUMBER: 1998:42085 USPATFULL

TITLE: Transdermal drug delivery device for treating erectile
dysfunction

INVENTOR(S): Lee, Hun Han, Taejeon, Korea, Republic of
Cho, Joong Woong, Taejeon, Korea, Republic of
Kim, Choul Young, Taejeon, Korea, Republic of
Pai, Chaul Min, Taejeon, Korea, Republic of
Song, Jin Deog, Taejeon, Korea, Republic of
Park, Chul Min, Taejeon, Korea, Republic of
Yoon, Hye Jeong, Taejeon, Korea, Republic of
Yeo, Yoon, Taejeon, Korea, Republic of
Paick, Jae Seung, Seoul, Korea, Republic of
PATENT ASSIGNEE(S): Sam Yang Co., Ltd., Seoul, Korea, Republic of
(non-U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5741511	19980421
APPLICATION INFO.:	US 1996-627805	19960410 (8)

	NUMBER	DATE
PRIORITY INFORMATION:	KR 1995-8444	19950412
	KR 1995-43422	19951124
	KR 1996-6908	19960315

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Phelan, D. Gabrielle
LEGAL REPRESENTATIVE: Kenyon & Kenyon
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 445

AB The present invention is to provide a method and a transdermal drug
delivery device for treating erectile dysfunction which comprises a
patch containing pharmaceutically active ingredient and being directly
apply to the male glans penis and its support and the rings for
constricting the base part of the penis to aid the erection. The patch
according to the present invention may be divided into two types, i.e.
a cylinder type patch and a multi-reservoir type patch. The transdermal
drug delivery patch device of the present invention is painless and
safely to use and may be effectively produced and maintained erection
of the penis without the adverse side effects and with a high degree of
patient acceptability in the case of male impotence.

=> d kwic

L12 ANSWER 1 OF 1 USPATFULL

SUMM . . . a condom may cause the side effects including allergy due to the condom to the male as well as the **female**, and may not give the **orgasm** during the **sexual** intercourse. Therefore, such patch system may not apply to the persons having the rejection symptoms on the use of condom.

DETD Diltiazem, nifedipine, nicardipine, nimodipine and/or verapamil can be used as calcium blockers. Further, PGE1, alprostadil, **misoprostol** and/or PGE2 as natural and synthetic vasoactive prostaglandins; apomorphine, bromocriptine as dopamine antagonists; adenosine, amylnitrate, dipyridamole, erythrityl tetranitrate, ethaverine hydrochloride, . . .

=> fil stng

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	5.72	50.37
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'STNGUIDE' ENTERED AT 12:00:21 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

=> index bioscience

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.00	50.37
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCERMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:02:52 ON 14 MAY 2001

59 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> fil medline embase biosis uspatfull

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.45	50.82

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'MEDLINE' ENTERED AT 12:03:09 ON 14 MAY 2001

FILE 'EMBASE' ENTERED AT 12:03:09 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 12:03:09 ON 14 MAY 2001
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'USPATFULL' ENTERED AT 12:03:09 ON 14 MAY 2001
CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

=> s l1 or misoprostol or (prostaglandin E) or PGE
L13 23010 L1 OR MISOPROSTOL OR (PROSTAGLANDIN E) OR PGE

=> s l13 and l10
L14 55 L13 AND L10

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15 41 DUP REM L14 (14 DUPLICATES REMOVED)

=> s l13 (s) l10
L16 37 L13 (S) L10

=> dup rem l16
PROCESSING COMPLETED FOR L16
L17 23 DUP REM L16 (14 DUPLICATES REMOVED)

=> d ibib abs 1-10

L17 ANSWER 1 OF 23 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001026433 EMBASE
TITLE: Fetal androgen exposure inhibits fetal rat lung fibroblast lipid uptake and release.
AUTHOR: Rodriguez A.; Viscardi R.M.; Torday J.S.
CORPORATE SOURCE: Dr. A. Rodriguez, Department of Pediatrics, Mercy Medical Center, 301 St. Paul's Place, Baltimore, MD 21202, United States. andres2@home.com
SOURCE: Experimental Lung Research, (2001) 27/1 (13-24).
Refs: 30
ISSN: 0190-2148 CODEN: EXLRDA
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
021 Developmental Biology and Teratology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Fetal lung fibroblasts provide lipid substrate for type II cell surfactant phospholipid synthesis. This process is developmental and glucocorticoid dependent. Previous studies in our laboratory demonstrating **sex** differences in several aspects of lung maturation suggest that these differences may be due to effects of fetal androgens. Based on these

studies, we hypothesized that fetal lung fibroblast triglyceride metabolism is determined by opposing effects of fetal androgens and glucocorticoids. To model the effects of androgens on fetal lung fibroblast triglyceride metabolism, pregnant rats were treated with dihydrotestosterone (DHT) 1 mg/kg/day from the days 15 to 20 of gestation, and changes in triglyceride content of freshly isolated fetal rat lung fibroblasts (FRLF) and rates of uptake and prostaglandin F(2) (PGE (2))-mediated release by cultured FRLF in **response** to glucocorticoids in the presence or absence of DHT in vitro were measured. During lung development, the triglyceride content and rate of uptake of **female**-derived FRLF increased 3.5- and 4.8-fold, respectively, between days 18 and 20 of gestation. From days 19 to 22, male FRLF triglyceride content and rate of uptake were lower than the content and uptake by **female** FRLF. Maternal DHT treatment inhibited the normal developmental increase in fibroblast triglyceride content and rate of uptake between days 19 and 22 by both male and **female** FRLF. In the absence of maternal DHT, in vitro dexamethasone stimulated triglyceride uptake 3-fold by day 21 in FRLF. This effect was blocked by maternal pretreatment with DHT. Maternal DHT exposure prevented stimulation of triglyceride release by PGF(2). Although in vitro dexamethasone stimulated triglyceride release by maternal DHT-exposed fibroblasts, it did not enhance the **response** to PGE (2). These data suggest that in utero exposure to androgens (1) delay the developmental increase in triglyceride content and (2) oppose the effects of glucocorticoid on cultured FRLF triglyceride uptake and PGE (2)-mediated release.

L17 ANSWER 2 OF 23 MEDLINE

ACCESSION NUMBER: 2000027129 MEDLINE

DOCUMENT NUMBER: 20027129 PubMed ID: 10559400

TITLE: Neuregulins signaling via a glial erbB-2-erbB-4 receptor complex contribute to the neuroendocrine control of mammalian sexual development.

AUTHOR: Ma Y J; Hill D F; Creswick K E; Costa M E; Córnea A; Lioubin M N; Plowman G D; Ojeda S R

CORPORATE SOURCE: Division of Neuroscience, Oregon Regional Primate Research Center, Beaverton, Oregon 97006, USA.

CONTRACT NUMBER: HD25123 (NICHD)
P30 HD18185 (NICHD)
RR00163 (NCRR)

SOURCE: JOURNAL OF NEUROSCIENCE, (1999 Nov 15) 19 (22) 9913-27.
Journal code: JDF; 8102140. ISSN: 0270-6474.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199912

ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20000113
Entered Medline: 19991202

AB Activation of erbB-1 receptors by glial TGFalpha has been shown to be a component of the developmental program by which the neuroendocrine brain controls mammalian **sexual** development. The participation of other members of the erbB family may be required, however, for full signaling capacity. Here, we show that activation of astrocytic erbB-2/erbB-4 receptors plays a significant role in the process by which the hypothalamus controls the advent of mammalian **sexual** maturation. Hypothalamic astrocytes express both the erbB-2 and erbB-4 genes, but no erbB-3, and **respond** to neuregulins (NRGs) by

releasing prostaglandin E(2) (PGE(2)), which acts on neurosecretory neurons to stimulate secretion of luteinizing hormone-releasing hormone (LHRH), the neuropeptide controlling **sexual** development. The actions of TGFalpha and NRGs in glia are synergistic and involve recruitment of erbB-2 as a coreceptor, via erbB-1 and erbB-4, respectively. Hypothalamic expression of both erbB-2 and erbB-4 increases first in a gonad-independent manner before the onset of puberty, and then, at the time of puberty, in a **sex** steroid-dependent manner. Disruption of erbB-2 synthesis in hypothalamic astrocytes by treatment with an antisense oligodeoxynucleotide inhibited the astrocytic **response** to NRGs and, to a lesser extent, that to TGFalpha and blocked the erbB-dependent, glia-mediated, stimulation of LHRH release. Intracerebral administration of the oligodeoxynucleotide to developing animals delayed the initiation of puberty. Thus, activation of the erbB-2-erbB-4 receptor complex appears to be a critical component of the signaling process by which astrocytes facilitate the acquisition of **female** reproductive capacity in mammals.

L17 ANSWER 3 OF 23 MEDLINE
 ACCESSION NUMBER: 1999384152 MEDLINE
 DOCUMENT NUMBER: 99384152 PubMed ID: 10453054
 TITLE: Glial-neuronal interactions in the neuroendocrine control of mammalian puberty: facilitatory effects of gonadal steroids.
 AUTHOR: Ojeda S R; Ma Y J
 CORPORATE SOURCE: Division of Neuroscience, Oregon Regional Primate Research Center/Oregon Health Sciences University, 505 N.W. 185th Avenue, Beaverton, Oregon 97006, USA.
 CONTRACT NUMBER: HD25123 (NICHD)
 P30 HD18185 (NICHD)
 RR00163 (NCRR)
 SOURCE: JOURNAL OF NEUROBIOLOGY, (1999 Sep 15) 40 (4) 528-40.
 Ref: 95
 Journal code: JAM; 0213640. ISSN: 0022-3034.
 PUB. COUNTRY: United States
 Journal; Article; (JOURNAL ARTICLE)
 General Review; (REVIEW)
 (REVIEW, TUTORIAL)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199910
 ENTRY DATE: Entered STN: 19991014
 Last Updated on STN: 19991014
 Entered Medline: 19991004
 AB It is now clear that astroglial cells actively contribute to both the generation and flow of information within the central nervous system. In the hypothalamus, astrocytes regulate the secretory activity of neuroendocrine neurons. A small subset of these neurons secrete luteinizing hormone-releasing hormone (LHRH), a neuropeptide essential for **sexual** development and adult reproductive function. Astrocytes stimulate LHRH secretion via cell-cell signaling mechanisms involving growth factors recognized by receptors with either serine/threonine or tyrosine kinase activity. Two members of the epidermal growth factor (EGF) family and their respective tyrosine kinase receptors appear to play key roles in this regulatory process. Transforming growth factor-alpha (TGFalpha) and its distant congeners, the neuregulins (NRGs), are produced

in hypothalamic astrocytes. They stimulate LHRH secretion indirectly, via activation of erbB-1/erbB-2 and erbB-4/erbB-2 receptor complexes also located on astrocytes. Activation of these receptors leads to release of prostaglandin E(2) (PGE(2)), which then binds to specific receptors on LHRH neurons to elicit LHRH secretion. Gonadal steroids facilitate this glia-to-neuron communication process by acting at three different steps along the signaling pathway. They (a) increase astrocytic gene expression of at least one of the EGF-related ligands (TGFalpha),

- (b) increase expression of at least two of the receptors (erbB-4 and erbB-2), and (c) enhance the LHRH **response** to PGE(2) by up-regulating in LHRH neurons the expression of specific PGE(2) receptor isoforms. Focal overexpression of TGFalpha in either the median eminence or preoptic area of the hypothalamus accelerates puberty. Conversely, blockade of either TGFalpha or NRG hypothalamic actions

delays

the process. Thus, both TGFalpha and NRGs appear to be physiological components of the central neuroendocrine mechanism controlling the initiation of **female** puberty. By facilitating growth factor signaling pathways in the hypothalamus, ovarian steroids accelerate the pace and progression of the pubertal process.
Copyright 1999 John Wiley & Sons, Inc.

L17 ANSWER 4 OF 23 MEDLINE DUPLICATE 1
 ACCESSION NUMBER: 1999264150 MEDLINE
 DOCUMENT NUMBER: 99264150 PubMed ID: 10333355
 TITLE: Seminal plasma components stimulate interleukin-8 and interleukin-10 release.
 AUTHOR: Denison F C; Grant V E; Calder A A; Kelly R W
 CORPORATE SOURCE: Department of Obstetrics and Gynaecology, University of Edinburgh, Centre for Reproductive Biology, UK.
 SOURCE: MOLECULAR HUMAN REPRODUCTION, (1999 Mar) 5 (3) 220-6.
 Journal code: CWO; 9513710. ISSN: 1360-9947.
 PUB. COUNTRY: ENGLAND: United Kingdom

 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199907
 ENTRY DATE: Entered STN: 19990806
 Last Updated on STN: 19990806
 Entered Medline: 19990723

AB Human seminal plasma has potent anti-inflammatory properties which are thought to confer a survival advantage to the spermatozoa within the hostile **female genital** tract. In contrast, a profound pro-inflammatory leukocytosis has been observed post-coitus in animals

and

humans. Whether components of seminal plasma are involved in initiating this leukocytic reaction is not known. This study investigated the effect of human seminal plasma, a seminal plasma fraction and its principal constituent prostaglandins, prostaglandin E2 (PGE2) and 19-hydroxy PGE, on the release of the pro-inflammatory neutrophil chemotactic factor interleukin-8 (IL-8) and the anti-inflammatory cytokines interleukin-10 (IL-10) and secretory leukocyte protease inhibitor (SLPI). The tissues studied were non-pregnant cervical explants, peripheral blood and the monocyte cell line U937. Seminal plasma fraction (SPF) significantly (P < 0.05) stimulated release of IL-8 and inhibited release of SLPI from non-pregnant cervical explants. SPF, PGE2 and 19-hydroxy PGE significantly (P < 0.005) stimulated IL-8 release from peripheral blood and U937 cells. 19-hydroxy PGE was significantly (P < 0.005) more effective than PGE2 in stimulating IL-8

release. Seminal plasma, SPF and PGE2 significantly ($P < 0.05$) stimulated IL-10 release from U937 cells. 19-hydroxy **PGE** stimulated IL-10 release from U937 cells but this failed to reach significance. Release of IL-10 by cervical explants and SLPI by peripheral blood and U937 cells were below the detection limit of the assays employed. We suggest that the anti- and pro-inflammatory immune **responses** which seminal plasma induces might act in combination initially to promote sperm survival and then to facilitate their removal from the **female genital tract**.

L17 ANSWER 5 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:299489 BIOSIS
DOCUMENT NUMBER: PREV199799598692
TITLE: Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE-2 fever.
AUTHOR(S): Chen, X. (1); Landgraf, R.; Pittman, Q. J.
CORPORATE SOURCE: (1) Neuroscience Res. Group, Dep. Physiol. Biophysics, Univ. Calgary, 3330 Hospital Dr. NW, Calgary, AB T2N 4N1 Canada
SOURCE: American Journal of Physiology, (1997) Vol. 272, No. 5
PART 2, pp. R1664-R1669.
ISSN: 0002-9513.
DOCUMENT TYPE: Article
LANGUAGE: English

AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses**. We therefore examined the temperature **responses** of conscious and urethan-anesthetized rats of both **sexes** to centrally administered prostaglandin E-2 (**PGE-2**) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious (25 ng/5 μ l **PGE-2** intracerebroventricularly (icv)) and anesthetized (VSA microdialyzed, 50 ng/5 μ l **PGE-2** icv) **female** rats had higher fevers than did males. Infusion of an AVP V-1a receptor antagonist (1 nmol (d(CH-2)-5Tyr(Me))AVP) plus **PGE-2** gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to **PGE-2** in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in **PGE-2** fever, which may be accounted for by the differential AVP release in the VSA.

L17 ANSWER 6 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1997:449248 BIOSIS
DOCUMENT NUMBER: PREV199799748451
TITLE: Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae.
AUTHOR(S): Cheng, Ming Zhao; Zaman, Gul; Rawlinson, Simon C. F.; Fitsillides, Andrew A.; Suswillo, Rosemary F. L.; Lanyon, Lance E. (1)
CORPORATE SOURCE: (1) Royal Vet. College, Univ. London, Royal College St., London NW1 0TU UK
SOURCE: Journal of Bone and Mineral Research, (1997) Vol. 12, No.

9, pp. 1424-1430.

ISSN: 0884-0431.

DOCUMENT TYPE: Article

LANGUAGE: English

AB Explants of ulnae from 5-week-old male and **female** rats were cleaned of marrow and soft tissue and, in the presence and absence of 10-8

M 17-beta-estradiol (E2) or 5-alpha-dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 mu-epsilon, 1 Hz, 8 minutes, maximum strain rate 25,000 mu-epsilon/s), prostaglandin E-2 (**PGE-2**) and prostacyclin (PGI-2) (10-6 M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from males and **females**, as indicated by incorporation of (3H)thymidine into DNA and (3H)proline into collagen. E2 and DHT both increased (3H)thymidine

and (3H)proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **response** in (3H)thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and **PGE-2** or PGI-2. The interaction between loading and E-2 for (3H)proline incorporation was not mimicked by these prostanoids. In males the synergism in (3H) proline incorporation seen between loading and DHT was mimicked by that between PGI-2 and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**. Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are reproduced in some but not all cases by E-2 and prostaglandins. E-2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.

L17 ANSWER 7 OF 23 MEDLINE

DUPLICATE 2

ACCESSION NUMBER: 97302992 MEDLINE

DOCUMENT NUMBER: 97302992 PubMed ID: 9159423

TITLE: A cytokine switch induced by human seminal plasma: an immune modulation with implications for sexually transmitted disease.

AUTHOR: Kelly R W; Carr G G; Critchley H O

CORPORATE SOURCE: Medical Research Council Reproductive Biology Unit, University of Edinburgh Centre for Reproductive Biology, UK.

SOURCE: HUMAN REPRODUCTION, (1997 Apr) 12 (4) 677-81. Journal code: HRP; 8701199. ISSN: 0268-1161.

PUB. COUNTRY: ENGLAND: United Kingdom (CLINICAL TRIAL)

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199707

ENTRY DATE: Entered STN: 19970805

Last Updated on STN: 19970805

Entered Medline: 19970722

AB The immunosuppressive activity of human seminal plasma may be one factor in the aetiology of **sexually** transmitted disease and could be particularly important for the spread of human immunodeficiency virus (HIV). The advent of virus that can preferentially infect Langerhans cells

of the **genital** mucosa underscores the relevance of seminal plasma effects. Virally infected cells are eradicated by the killing activity of T cells and natural killer (NK) cells and this cytotoxicity

is

stimulated by IL-12 (previously known as natural killer cell stimulatory factor) and partly inhibited by IL-10 (previously known as cytokine synthesis inhibitory factor). We have examined the effects of human seminal plasma on the production of these key cytokines. Cytokine production was measured in rapidly diluted, fresh, lipopolysaccharide (LPS)-stimulated, whole blood since this provided leukocytes with minimal exposure to prostaglandin. Prostaglandin concentrations and cytokine release were measured by ELISA. Addition of human seminal plasma diluted up to 100,000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio ($P < 0.02$). A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma). This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (**PGE**) and 19-OH **PGE** (the main prostaglandin constituent of human seminal plasma). Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution. However, its activity could be restored by the addition of synthetic **PGE** and 19-hydroxy **PGE**. A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower **female** reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells. In addition, the

stimulation

of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells. The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower **genital** tract, with ablation of cell-mediated **responses** and immunosurveillance.

L17 ANSWER 8 OF 23 MEDLINE

DUPLICATE 3

ACCESSION NUMBER: 1998041214 MEDLINE

DOCUMENT NUMBER: 98041214 PubMed ID: 9373880

TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women.

AUTHOR: Farker K; Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth

H W; Hoffmann A; Oettel M

CORPORATE SOURCE: Institute of Clinical Pharmacology, Friedrich Schiller University, Jena, Germany.

SOURCE: PROTAGLANDINS, (1997 Sep) 54 (3) 655-64.
Journal code: Q76; 0320271. ISSN: 0090-6980.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199712

ENTRY DATE: Entered STN: 19980109

Last Updated on STN: 19980109

Entered Medline: 19971218

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand, and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E2 (PGE2), 6-keto-PGF1 alpha, thromboxane B2 (TxB2) and their metabolites **PGE-M** (11 alpha-hydroxy-9, 15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanic acid), 2,3-dinor-6-keto-PGF1 alpha, 2,3-dinor-TxB2 and 11-dehydro-TxB2 were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young non-pregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, PGE2 and TxB2 were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and postmenopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L17 ANSWER 9 OF 23 MEDLINE

DUPLICATE 4

ACCESSION NUMBER: 97115452 MEDLINE

DOCUMENT NUMBER: 97115452 PubMed ID: 8956853

TITLE: Self-reported sexual function and sexual arousability in women with epilepsy.

AUTHOR: Morrell M J; Guldner G T

CORPORATE-SOURCE: Department of Neurology and Neurological Sciences, Stanford

Comprehensive Epilepsy Center, Stanford University School of Medicine, California, USA.

SOURCE: EPILEPSIA, (1996 Dec) 37 (12) 1204-10.

Journal code: EIX; 2983306R. ISSN: 0013-9580.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199701

ENTRY DATE: Entered STN: 19970128

Last Updated on STN: 19990129

Entered Medline: 19970116

AB PURPOSE: **Women** with epilepsy are at risk for **sexual** dysfunction but the frequency and types of dysfunction have not been well characterized. METHODS: Self-reported **sexual** function was evaluated in 116 **women** aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17 with primary generalized epilepsy (**PGE**). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure. Standardized inventories assessed **sexual** functioning, **sexual** arousability and anxiety, **sexual** behavior, and depression. RESULTS: Although **sexual** experience was not reduced, **women** with **PGE** and LRE reported significantly less

sexual arousability and **women** with LRE reported significantly more **sexual** anxiety. **Women** with LRE experienced significantly more dyspareunia, vaginismus, **arousal** insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset. CONCLUSIONS: In contrast to subjects of previous research, the **women** in our study did not have a disorder of **sexual desire**, but more than one third experienced disorders of **sexual arousal**, implying a physiological deficit. Although the etiology for these **arousal** phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed in the treatment of **sexual** disorders.

L17 ANSWER 10 OF 23 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1996:360368 BIOSIS

DOCUMENT NUMBER: PREV199699082724

TITLE: Progesterone and dexamethasone stimulate proliferation and differentiation of osteoprogenitors and progenitors for adipocytes and macrophages in cell populations derived from

adult rat vertebrae.

AUTHOR(S): Ishida, Yoichiro (1); Tertinegg, Inka; Heersche, Johan N. M.

CORPORATE SOURCE: (1) Fac. Dentistry, Univ. Toronto, Room 400, 124 Edward Street, Toronto, ON M5G 1G6 Canada

SOURCE: Journal of Bone and Mineral Research, (1996) Vol. 11, No. 7, pp. 921-930.
ISSN: 0884-0431.

DOCUMENT TYPE: Article

LANGUAGE: English

AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in alpha-minimum essential medium plus 10% fetal calf serum (standard medium) and then subculturing the outgrowth cells in standard medium plus 50 μ -g/ml of ascorbic acid, 5 mM beta-glycerophosphate, and with or without Prog or Dex. On day 6 of culture, these populations were analyzed for cAMP **responses** to parathyroid hormone (PTH), prostaglandin E-2 (**PGE**-2), and isoproterenol (IPT). Increases in intracellular cAMP were seen in **response** to PTH, **PGE**-2, and IPT, and culturing in medium containing Prog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Prog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Prog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Prog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of alpha-naphthyl butyrate esterase (alpha-NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED-2-positive colonies (macrophages). Prog-treated cultures contained more colonies of small

spindle-shaped alpha-NBE-positive cells and fewer colonies of small round a-NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Prog and Dex. The data also suggest that the effects of Prog and Dex differ qualitatively and quantitatively.

=> s l17 range=, 1999
L18 21 L17

=> s l17 range=, 1998
L19 19 L17

=> d ti so tot

L19 ANSWER 1 OF 19 MEDLINE
TI Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women.
SO PROSTAGLANDINS, (1997 Sep) 54 (3) 655-64.
Journal code: Q76; 0320271. ISSN: 0090-6980.

L19 ANSWER 2 OF 19 MEDLINE
TI A cytokine switch induced by human seminal plasma: an immune modulation with implications for sexually transmitted disease.
SO HUMAN REPRODUCTION, (1997 Apr) 12 (4) 677-81.
Journal code: HRP; 8701199. ISSN: 0268-1161.

L19 ANSWER 3 OF 19 MEDLINE
TI Self-reported sexual function and sexual arousability in women with epilepsy.
SO EPILEPSIA, (1996 Dec) 37 (12) 1204-10.
Journal code: EIX; 2983306R. ISSN: 0013-9580.

L19 ANSWER 4 OF 19 MEDLINE
TI [Prostaglandins and reproduction. I. Physiological aspects].
Prostaglandines et reproduction. I. Aspects physiologiques.
SO JOURNAL DE GYNECOLOGIE, OBSTETRIQUE ET BIOLOGIE DE LA REPRODUCTION, (1991) 20 (1) 7-17. Ref: 69
Journal code: IAZ; 0322206. ISSN: 0368-2315.

L19 ANSWER 5 OF 19 MEDLINE
TI Effects of female sex hormones and pregnancy on gallbladder prostaglandin synthesis.
SO ARCHIVES OF SURGERY, (1988 Jun) 123 (6) 705-8.
Journal code: 8IA; 9716528. ISSN: 0004-0010.

L19 ANSWER 6 OF 19 MEDLINE
TI Prostaglandin-mediated inhibition of lymphokine secretion in normal individuals and patients with progressive systemic sclerosis (scleroderma, PSS).
SO AGENTS AND ACTIONS, (1982 Oct) 12 (4) 471-7.
Journal code: 2XZ; 0213341. ISSN: 0065-4299.

L19 ANSWER 7 OF 19 MEDLINE
TI How important are prostaglandins in the urology of man?.

- SO UROLOGIA INTERNATIONALIS, (1982) 37 (3) 160-71.
Journal code: WRI; 0417373. ISSN: 0042-1138.
- L19 ANSWER 8 OF 19 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
TI Effect of adrenoceptor blockers and maoi on postcoital utero-oviductal
contractility, sperm transport, and sperm attachment to eggs in rabbits.
SO Archives of Andrology, (1981) 6/4 (307-316).
CODEN: ARANDR
- L19 ANSWER 9 OF 19 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
TI Effects of cannabinoids and female exposure on the pituitary testicular
axis in mice: Possible involvement of prostaglandins.
SO Biology of Reproduction, (1981) 24/2 (315-322).
CODEN: BIREBV
- L19 ANSWER 10 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Enhancement by sex hormones of the osteoregulatory effects of mechanical
loading and prostaglandins in explants of rat ulnae.
SO Journal of Bone and Mineral Research, (1997) Vol. 12, No. 9, pp.
1424-1430.
ISSN: 0884-0431.
- L19 ANSWER 11 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Differential ventral septal vasopressin release is associated with sexual
dimorphism in PGE-2 fever.
SO American Journal of Physiology, (1997) Vol. 272, No. 5 PART 2, pp.
R1664-R1669.
ISSN: 0002-9513.
- L19 ANSWER 12 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Progesterone and dexamethasone stimulate proliferation and
differentiation
of osteoprogenitors and progenitors for adipocytes and macrophages in
cell
~~populations derived from adult rat vertebrae~~
SO Journal of Bone and Mineral Research, (1996) Vol. 11, No. 7, pp.
921-930.
ISSN: 0884-0431.
- L19 ANSWER 13 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Influence of the estrous cycle on the norepinephrine-induced contraction
of rat aorta: Relationship to vascular prostanoids biosynthesis.
SO Biological Research, (1994) Vol. 27, No. 3-4, pp. 209-215.
ISSN: 0716-9760.
- L19 ANSWER 14 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Mesenteric vascular responses to vasopressin during development of
DOCA-salt hypertension in male and female rats.
SO American Journal of Physiology, (1995) Vol. 268, No. 1 PART 2, pp.
R40-R49.
ISSN: 0002-9513.
- L19 ANSWER 15 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI Gender-related response to a tert-butyl hydroperoxide-induced oxidation
in
human neonatal tissue.
SO Free Radical Biology & Medicine, (1994) Vol. 16, No. 3, pp. 307-313.
ISSN: 0891-5849.
- L19 ANSWER 16 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS

TI Sexually dimorphic effects of alcohol exposure in utero on neuroendocrine
and immune functions in chronic alcohol-exposed adult rats.
SO Molecular and Cellular Neuroscience, (1993) Vol. 4, No. 4, pp. 343-353.
ISSN: 1044-7431.

L19 ANSWER 17 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI ROLE OF PROSTAGLANDINS IN FISH REPRODUCTION.
SO CAN J FISH AQUAT SCI, (1982) 39 (1), 92-98.
CODEN: CJFSDX. ISSN: 0706-652X.

L19 ANSWER 18 OF 19 BIOSIS COPYRIGHT 2001 BIOSIS
TI INFLUENCE OF SEX AND AGE ON FEBRILE RESPONSES TO PERIPHERAL AND CENTRAL
ADMINISTRATION OF PYROGENS IN THE RABBIT.
SO J PHYSIOL (LOND), (1979) 295 (0), 263-272.
CODEN: JPHYA7. ISSN: 0022-3751.

L19 ANSWER 19 OF 19 USPATFULL
TI Synergistic composition comprising PGF.sub.2.sub..alpha. and PGE.sub.2

=> fil stng

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	43.70	94.52
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'STNGUIDE' ENTERED AT 12:16:42 ON 14 MAY 2001
USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May 11, 2001 (20010511/UP).

=> index bioscience

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.00	94.52
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS,
BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT,
CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE,
DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:20:12 ON 14
MAY 2001

59 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s 11 or misoprostol or (prostagladin E) or PGE
1241* FILE ADISALERTS

39 FILE ADISINSIGHT
 81 FILE AGRICOLA
 24 FILE ANABSTR
 104* FILE AQUASCI
 172 FILE BIOBUSINESS
 2* FILE BIOCOMMERCE
 9147 FILE BIOSIS
 15 FILE BIOTECHABS
 15 FILE BIOTECHDS
 4490 FILE BIOTECHNO
 439* FILE CABA
 808 FILE CANCERLIT
 5060* FILE CAPLUS
 35* FILE CEABA-VTB
 1 FILE CEN
 76 FILE CIN
 361* FILE CONFSCI
 0* FILE CROPB
 2* FILE CROPU
 120* FILE DDFB
 2488* FILE DDFU
 22 FILES SEARCHED...
 61* FILE DGENE
 120* FILE DRUGB
 121 FILE DRUGLAUNCH
 125 FILE DRUGMONOG2
 19 FILE DRUGNL
 2874* FILE DRUGU
 14 FILE DRUGUPDATES
 171* FILE EMBAL
 5933 FILE EMBASE
 3570* FILE ESBIODBASE
 0* FILE FOMAD
 0* FILE FOREGE
 11* FILE FROSTI
 14 FILE FSTA
 25* FILE GENBANK
 26* FILE HEALSAFE
 259* FILE IFIPAT
 282 FILE JICST-EPLUS
 3* FILE KOSMET
 3307* FILE LIFESCI
 1* FILE MEDICONF
 43 FILES SEARCHED...
 4475 FILE MEDLINE
 45 FILE NIOSHTIC
 84* FILE NTIS
 23* FILE OCEAN
 5188* FILE PASCAL
 30 FILE PHAR
 0* FILE PHIC
 476* FILE PHIN
 1371 FILE PROMT
 8868* FILE SCISEARCH
 6 FILE SYNTHLINE
 1693 FILE TOXLINE
 2279 FILE TOXLIT
 3435* FILE USPATFULL
 375 FILE WPIDS
 58 FILES SEARCHED...

375 FILE WPINDEX

55 FILES HAVE ONE OR MORE ANSWERS, 59 FILES SEARCHED IN STNINDEX

L20 QUE L1 OR MISOPROSTOL OR (PROSTAGLADIN E) OR PGE

=> s (female or woman or women or girl or lady) (s) ((sex? or genital) (s)
(hypoactiv? or desir? or satisfact? or orgasm or arousal? or sensation? or
respon? or anhedonia))

283 FILE ADISALERTS
69 FILE ADISINSIGHT
832 FILE AGRICOLA
2 FILE ANABSTR
734 FILE AQUASCI
303 FILE BIOBUSINESS
2 FILE BIOCOMMERCE
15038 FILE BIOSIS
12 FILE BIOTECHABS
9 FILES SEARCHED...
12 FILE BIOTECHDS
1825 FILE BIOTECHNO
4136 FILE CABA
2254 FILE CANCERLIT
13 FILES SEARCHED...
1933 FILE CAPLUS
2 FILE CEABA-VTB
11 FILE CEN
9 FILE CIN
23 FILE CONFSCI
117 FILE CROPB
374 FILE CROPU
96 FILE DDFB
421 FILE DDFU
22 FILES SEARCHED...
100 FILE DGENE
96 FILE DRUGB
8 FILE DRUGNL
1532 FILE DRUGU
14 FILE DRUGUPDATES
152 FILE EMBAL
13934 FILE EMBASE
31 FILES SEARCHED...
3509 FILE ESBIODASE
2 FILE FOMAD
21 FILE FROSTI
61 FILE FSTA
1 FILE GENBANK
37 FILES SEARCHED...
231 FILE HEALSAFE
57 FILE IFIPAT
748 FILE JICST-EPLUS
15 FILE KOSMET
4182 FILE LIFESCI
2 FILE MEDICONF
13531 FILE MEDLINE
44 FILES SEARCHED...
561 FILE NIOSHTIC
347 FILE NTIS
185 FILE OCEAN
4100 FILE PASCAL

9 FILE PHAR
49 FILES SEARCHED...
1 FILE PHIC
64 FILE PHIN
1233 FILE PROMT
7404 FILE SCISEARCH
3855 FILE TOXLINE
55 FILES SEARCHED...
3780 FILE TOXLIT
943 FILE USPATFULL
91 FILE WPIDS
91 FILE WPINDEX

55 FILES HAVE ONE OR MORE ANSWERS, 59 FILES SEARCHED IN STNINDEX

L21 QUE (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR GENITAL)
(S)
(HYPOACTIV? OR DESIR? OR SATISFACT? OR ORGASM OR AROUSAL? OR
SENSATION
? OR RESPON? OR ANHEDONIA))

=> s 120 and 121

1* FILE ADISALERTS
1 FILE ADISINSIGHT
0* FILE AQUASCI
0* FILE BIOCOMMERCE
19 FILE BIOSIS
10 FILES SEARCHED...
8 FILE BIOTECHNO
2* FILE CABA
4 FILE CANCERLIT
7* FILE CAPLUS
14 FILES SEARCHED...
0* FILE CEABA-VTB
~~0* FILE CONFSCI~~
0* FILE CROPB
0* FILE CROPU
0* FILE DDFB
1* FILE DDFU
0* FILE DGENE
0* FILE DRUGB
3* FILE DRUGU
1 FILE DRUGUPDATES
29 FILES SEARCHED...
0* FILE EMBAL
11 FILE EMBASE
8* FILE ESBIODBASE
0* FILE FOMAD
0* FILE FOREGE
0* FILE FROSTI
0* FILE GENBANK
37 FILES SEARCHED...
0* FILE HEALSAFE
0* FILE IFIPAT
0* FILE KOSMET
6* FILE LIFESCI
0* FILE MEDICONF
10 FILE MEDLINE
45 FILES SEARCHED...
0* FILE NTIS

0* FILE OCEAN
 10* FILE PASCAL
 0* FILE PHIC
 0* FILE PHIN
 5 FILE PROMT
 52 FILES SEARCHED...
 18* FILE SCISEARCH
 2 FILE TOXLINE
 8 FILE TOXLIT
 15* FILE USPATFULL
 57 FILES SEARCHED...
 1 FILE WPIDS
 1 FILE WPINDEX

22 FILES HAVE ONE OR MORE ANSWERS, 59 FILES SEARCHED IN STNINDEX

L22 QUE L20 AND L21

=> d rank

F1	19	BIOSIS
F2	18*	SCISEARCH
F3	15*	USPATFULL
F4	11	EMBASE
F5	10	MEDLINE
F6	10*	PASCAL
F7	8	BIOTECHNO
F8	8	TOXLIT
F9	8*	ESBIOBASE
F10	7*	CAPLUS
F11	6*	LIFESCI
F12	5	PROMT
F13	4	CANCERLIT
F14	3*	DRUGU
F15	2	TOXLINE
F16	2*	CABA
F17	1	ADISINSIGHT
F18	1	DRUGUPDATES
F19	1	WPIDS
F20	1	WPINDEX
F21	1*	ADISALERTS
F22	1*	DDFU

=> file f2, f6-f9, f11-f22

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	5.40	99.92
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-0.59

FILE 'SCISEARCH' ENTERED AT 12:27:36 ON 14 MAY 2001
 COPYRIGHT (C) 2001 Institute for Scientific Information (ISI) (R)

FILE 'PASCAL' ENTERED AT 12:27:36 ON 14 MAY 2001
 Any reproduction or dissemination in part or in full,
 by means of any process and on any support whatsoever
 is prohibited without the prior written agreement of INIST-CNRS.
 COPYRIGHT (C) 2001 INIST-CNRS. All rights reserved.

FILE 'BIOTECHNO' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'TOXLIT' ENTERED AT 12:27:36 ON 14 MAY 2001

FILE 'ESBIOBASE' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'LIFESCI' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'PROMT' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Gale Group. All rights reserved.

FILE 'CANCERLIT' ENTERED AT 12:27:36 ON 14 MAY 2001

FILE 'DRUGU' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'TOXLINE' ENTERED AT 12:27:36 ON 14 MAY 2001

FILE 'CABA' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 CAB INTERNATIONAL (CABI)

FILE 'ADISINSIGHT' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'DRUGUPDATES' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 IMSWORLD Publications Ltd

FILE 'WPIDS' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'WPINDEX' ACCESS NOT AUTHORIZED

FILE 'ADISALERTS' ENTERED AT 12:27:36 ON 14 MAY 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'DDFU' ACCESS NOT AUTHORIZED

=> s f22
L23 605 F22

=> s 122
'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
 3 FILES SEARCHED...
'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
 7 FILES SEARCHED...
 10 FILES SEARCHED...
 14 FILES SEARCHED...
L24 79 L22

=> s 120 (s) 121
'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE
 3 FILES SEARCHED...

'CN' IS NOT A VALID FIELD CODE
'CN' IS NOT A VALID FIELD CODE

7 FILES SEARCHED...

9 FILES SEARCHED...

12 FILES SEARCHED...

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L65 (S) L66'
L25 69 L20 (S) L21

=> s cellulose
L26 222051 CELLULOSE

=> s l26 (s) l25
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'L129 (S) L113'
L27 0 L26 (S) L25

=> s l26 and l25
L28 0 L26 AND L25

=> d l25 1-5 ibib abs kwic

L25 ANSWER 1 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 2001:59328 SCISEARCH

THE GENUINE ARTICLE: 390QT

TITLE: Fetal androgen exposure inhibits fetal rat lung
fibroblast

lipid uptake and release
AUTHOR: Rodriguez A (Reprint); Viscardi R M; Torday J S
CORPORATE SOURCE: Mercy Med Ctr, Dept Pediat, 301 St Pauls Pl, Baltimore,
MD 21202 USA (Reprint); Univ Maryland, Sch Med, Dept Pediat,
Div Neonatol, Baltimore, MD 21201 USA

COUNTRY OF AUTHOR: USA

SOURCE: EXPERIMENTAL LUNG RESEARCH (JAN-FEB 2001) Vol. 27, No. 1,

pp. 13-24.

Publisher: HEMISPHERE PUBL CORP, 1900 FROST ROAD, SUITE
101, BRISTOL, PA 19007-1598 USA.

ISSN: 0190-2148.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 30

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Fetal lung fibroblasts provide lipid substrate for the II cell
surfactant phospholipid synthesis. This process is developmental and
glucocorticoid dependent. Previous studies in our laboratory
demonstrating

sex differences in several aspects of lung maturation suggest that
these differences may be due to effects of fetal androgens. Based on
these

studies, we hypothesized that fetal lung fibroblast triglyceride
metabolism is determined by opposing effects of fetal androgens and
glucocorticoids. To model the effects of androgens on fetal lung
fibroblast triglyceride metabolism, pregnant rats were treated with
dihydrotestosterone (DHT) 1 mg/kg/day from the days 15 to 20 of
gestation

and changes in triglyceride content of freshly isolated fetal rat lung
fibroblasts (FRLF) and rates of uptake and prostaglandin E-2 (PGE
(2))-mediated release by cultured FRLF in response to

glucocorticoids in the presence or absence of DHT In vitro were measured. During lung development, the triglyceride content and rate of uptake of **female**-derived FRLF increased 3.5- and 4.8-fold, respectively, between days 18 and 20 of gestation. From days 19 to 22, male FRLF triglyceride content and rate of uptake were lower than the content and uptake by **female** FRLF. Maternal DHT treatment inhibited the normal developmental increase in fibroblast triglyceride content and rate of uptake between days 19 and 22 by both male and **female** FRLF. In the absence of maternal DHT, in vitro dexamethasone stimulated triglyceride uptake 3-fold by day 21 in FRLF. This effect was blocked by maternal pre-treatment with DHT. Maternal DHT exposure prevented stimulation of triglyceride release by **PGE**(2). Although in vitro dexamethasone stimulated triglyceride release by maternal DHT-exposed fibroblasts, it did not enhance the **response** to **PGE**(2). These data suggest that in utero exposure to androgens (1) delay the developmental increase in triglyceride content and (2) oppose the effects of glucocorticoid on cultured FRLF triglyceride uptake and **PGE**(2)-mediated release.

AB . . . for the II cell surfactant phospholipid synthesis. This process is developmental and glucocorticoid dependent. Previous studies in our laboratory demonstrating **sex** differences in several aspects of lung maturation suggest that these differences may be due to effects of fetal androgens. Based . . . and changes in triglyceride content of freshly isolated fetal rat lung fibroblasts (FRLF) and rates of uptake and prostaglandin E-2 (**PGE**(2))-mediated release by cultured FRLF in **response** to glucocorticoids in the presence or absence of DHT In vitro were measured. During lung development, the triglyceride content and rate of uptake of **female**-derived FRLF increased 3.5- and 4.8-fold, respectively, between days 18 and 20 of gestation. From days 19 to 22, male FRLF triglyceride content and rate of uptake were lower than the content and uptake by **female** FRLF. Maternal DHT treatment inhibited the normal developmental increase in fibroblast triglyceride content and rate of uptake between days 19 and 22 by both male and **female** FRLF. In the absence of maternal DHT, in vitro dexamethasone stimulated triglyceride uptake 3-fold by day 21 in FRLF. This effect was blocked by maternal pre-treatment with DHT. Maternal DHT exposure prevented stimulation of triglyceride release by **PGE**(2). Although in vitro dexamethasone stimulated triglyceride release by maternal DHT-exposed fibroblasts, it did not enhance the **response** to **PGE**(2). These data suggest that in utero exposure to androgens (1) delay the developmental increase in triglyceride content and (2) oppose the effects of glucocorticoid on cultured FRLF triglyceride uptake and **PGE**(2)-mediated release.

L25 ANSWER 2 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
 ACCESSION NUMBER: 1999:941986 SCISEARCH
 THE GENUINE ARTICLE: 260YL
 TITLE: The immunobiology of sexual behavior: Gender differences in the suppression of sexual activity during illness
 AUTHOR: Avitsur R; Yirmiya R (Reprint)
 CORPORATE SOURCE: HEBREW UNIV JERUSALEM, DEPT PSYCHOL, IL-91905 JERUSALEM, ISRAEL (Reprint); HEBREW UNIV JERUSALEM, DEPT PSYCHOL, IL-91905 JERUSALEM, ISRAEL
 COUNTRY OF AUTHOR: ISRAEL

SOURCE: PHARMACOLOGY BIOCHEMISTRY AND BEHAVIOR, (DEC 1999) Vol. 64, No. 4, pp. 787-796.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
ISSN: 0091-3057.
DOCUMENT TYPE: General Review; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 117

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Following infection or injury, sick individuals experience profound psychological and behavioral changes, such as anorexia, depressed activity, and reduced self-care behavior. In the present review, we present evidence for a gender-difference in the behavioral **response** to sickness. Specifically, following immune activation, **sexual** activity is suppressed in **female**, but not in male rats. This gender difference is specific to **sexually** related **responses**, because other behaviors, such as locomotion, are equally affected by immune challenges in males and estrous **females**. The suppression of **female sexual** behavior, induced by either endotoxin (lipopolysaccharide), or the cytokine interleukin-1 (IL-1), are mediated by central mechanisms that are independent of alterations in ovarian hormone secretion. Furthermore, synergistic effects

of the cytokines IL-1 and tumor necrosis factor alpha (TNF alpha) are involved in modulating **sexual** behavior in sick **females**, and prostaglandins synthesis is required for the effects of IL-1 on **female sexual** behavior. The gender difference in the behavioral **response** to immune activation may be related to the findings that at the same doses and timing in which IL-1 suppressed **sexual** activity in **female** but not in male rats, **females** produced more prostaglandin E-2 (PGE(2)) in the brain, and less corticosterone than males. Finally, we are suggesting that

the suppressive effect of cytokines on **female** reproductive behavior may serve as a mechanism to reduce conception during infection, which exposes the mother and the fetus to dangers such as spontaneous abortions, preterm labor and maternal mortality. (C) 1999 Elsevier Science Inc.

AB . . . anorexia, depressed activity, and reduced self-care behavior. In the present review, we present evidence for a gender-difference in the behavioral **response** to sickness. Specifically, following immune activation, **sexual** activity is suppressed in **female**, but not in male rats. This gender difference is specific to **sexually** related **responses**, because other behaviors, such as locomotion, are equally affected by immune challenges in males and

estrous **females**. The suppression of **female sexual** behavior, induced by either endotoxin (lipopolysaccharide), or the cytokine interleukin-1 (IL-1), are mediated by central mechanisms that are independent of . . . hormone secretion. Furthermore, synergistic effects of the cytokines IL-1 and tumor necrosis factor alpha (TNF alpha) are involved in modulating **sexual** behavior in sick **females**, and prostaglandins synthesis is required for the effects of IL-1 on **female sexual** behavior. The gender difference in the behavioral **response** to immune activation may be related to the findings that at the same doses and timing in which IL-1 suppressed **sexual** activity in **female** but not in male

rats, **females** produced more prostaglandin E-2 (**PGE(2)**) in the brain, and less corticosterone than males. Finally, we are suggesting that the suppressive effect of cytokines on **female** reproductive behavior may serve as a mechanism to reduce conception during infection, which exposes the mother and the fetus to. . .

L25 ANSWER 3 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:879260 SCISEARCH
THE GENUINE ARTICLE: 254JG
TITLE: Neuregulins signaling via a glial erbB-2-erbB-4 receptor complex contribute to the neuroendocrine control of mammalian sexual development
AUTHOR: Ma Y J; Hill D F; Creswick K E; Costa M E; Cornea A; Lioubin M N; Plowman G D; Ojeda S R (Reprint)
CORPORATE SOURCE: OREGON REG PRIMATE RES CTR, DIV NEUROSCI, 505 NW 185TH AVE, BEAVERTON, OR 97006 (Reprint); OREGON REG PRIMATE RES CTR, DIV NEUROSCI, BEAVERTON, OR 97006; SUGEN INC, S SAN FRANCISCO, CA 94080
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF NEUROSCIENCE, (15 NOV 1999) Vol. 19, No. 22, pp. 9913-9927.
Publisher: SOC NEUROSCIENCE, 11 DUPONT CIRCLE, NW, STE 500, WASHINGTON, DC 20036.
ISSN: 0270-6474.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 77

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Activation of erbB-1 receptors by glial TGF alpha has been shown to be a component of the developmental program by which the neuroendocrine brain

~~controls mammalian sexual development.~~ The participation of other members of the erbB family may be required, however, for full signaling capacity. Here, we show that activation of astrocytic erbB-2/erbB-4 receptors plays a significant role in the process by which the hypothalamus controls the advent of mammalian **sexual** maturation. Hypothalamic astrocytes express both the erbB-2 and erbB-4 genes, but no erbB-3, and **respond** to neuregulins (NRGs) by releasing prostaglandin E-2 (**PGE(2)**), which acts on neurosecretory neurons to stimulate secretion of luteinizing hormone-releasing hormone (LHRH), the neuropeptide controlling **sexual** development. The actions of TGF alpha and NRGs in glia are synergistic and involve recruitment of erbB-2 as a coreceptor, via erbB-1 and erbB-4, respectively. Hypothalamic expression of both erbB-2 and erbB-4 increases first in a gonad-independent manner before the onset of puberty, and then, at the time of puberty, in a **sex** steroid-dependent manner. Disruption of erbB-2 synthesis in hypothalamic astrocytes by treatment with an antisense oligodeoxynucleotide inhibited the astrocytic **response** to NRGs and, to a lesser extent, that to TGF alpha and blocked the erbB-dependent, glia-mediated, stimulation of LHRH release. Intracerebral administration of the oligodeoxynucleotide to developing animals delayed the initiation of puberty. Thus, activation of the erbB-2-erbB-4 receptor complex appears to be a critical component of the signaling process by which astrocytes facilitate the acquisition of **female** reproductive capacity in mammals.

AB . . . TGF alpha has been shown to be a component of the developmental program by which the neuroendocrine brain controls mammalian

sexual development. The participation of other members of the erbB family may be required, however, for full signaling capacity. Here, we.

. of astrocytic erbB-2/erbB-4 receptors plays a significant role in the process by which the hypothalamus controls the advent of mammalian **sexual** maturation. Hypothalamic astrocytes express both the erbB-2 and erbB-4 genes, but no erbB-3, and **respond** to neuregulins (NRGs) by releasing prostaglandin E-2 (PGE(2)), which acts on neurosecretory neurons to stimulate secretion of luteinizing hormone-releasing hormone (LHRH), the neuropeptide controlling **sexual** development. The actions of TGF alpha and NRGs in glia are synergistic and involve recruitment of erbB-2 as a coreceptor, . . . increases first in a gonad-independent manner before the onset of puberty, and then, at the time of puberty, in a **sex** steroid-dependent manner. Disruption of erbB-2 synthesis in hypothalamic astrocytes by treatment with an antisense oligodeoxynucleotide inhibited the astrocytic **response** to NRGs and, to a lesser extent, that to TGF alpha and blocked the erbB-dependent, glia-mediated, stimulation of LHRH release. . . erbB-2-erbB-4 receptor complex appears to be a critical component of the signaling process by which astrocytes facilitate the acquisition of **female** reproductive capacity in mammals.

L25 ANSWER 4 OF 69 \SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 1999:682829 SCISEARCH

THE GENUINE ARTICLE: 231YE

TITLE: Identification of specific EP receptors responsible for the hemodynamic effects of PGE(2)

AUTHOR: Audoly L P; Tilley S L; Goulet J; Key M; Nguyen M; Stock J

CORPORATE SOURCE: L; McNeish J D; Koller B H; Coffman T M (Reprint)
DUKE UNIV, MED CTR, DIV NEPHROL, DEPT MED, BOX 3014, DURHAM, NC 27710 (Reprint); DUKE UNIV, MED CTR, DIV NEPHROL, DEPT MED, DURHAM, NC 27710; DURHAM VET AFFAIRS MED CTR, DURHAM, NC 27710; UNIV N CAROLINA, DEPT MED, CHAPEL HILL, NC 27599; PFIZER INC, CTR EXPT THERAPEUT, GROTON, CT 06340

COUNTRY OF AUTHOR: USA

SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY, (SEP 1999) Vol. 46, No. 3, pp. H924-H930. Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.

ISSN: 0363-6135.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 39

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To identify the E-prostanoid (EP) receptors that mediate the hemodynamic actions of PGE(2), we studied acute vascular **responses** to infusions of PGE(2) using lines of mice in which each of four EP receptors (EP1 through EP4) have been disrupted by gene targeting. In mixed groups of males and **females**, vasodepressor **responses** after infusions of PGE(2) were significantly diminished in the EP2 (-/-) and EP4 (-/-) lines but not in the EP1 -/- or EP3 -/- lines. Because the actions of other hormonal systems that regulate blood pressure differ between **sexes**, we compared the roles of individual EP receptors in males and **females**. We found that the relative contribution of each EP-receptor subclass

was

strikingly different in males from that in **females**. In **females**, the EP2 and EP4 receptors, which signal by stimulating adenylate cyclase, mediate the major portion of the vasodepressor **response** to PGE2. In males, the EP2 receptor has a modest effect, but most of the vasodepressor effect is mediated by the phospholipase C-coupled EP1 receptor. Finally in male mice, the EP3 receptor actively opposes the vasodepressor actions of **PGE(2)**. Thus the hemodynamic actions of **PGE(2)** are mediated through complex interactions of several EP-receptor subtypes, and the role of individual EP receptors differs dramatically in males from that in **females**. These differences may contribute to **sexual** dimorphism of blood pressure regulation.

AB To identify the E-prostanoid (EP) receptors that mediate the hemodynamic actions of **PGE(2)**, we studied acute vascular **responses** to infusions of **PGE(2)** using lines of mice in which each of four EP receptors (EP1 through EP4) have been disrupted by gene targeting. In mixed groups of males and **females**, vasodepressor **responses** after infusions of **PGE(2)** were significantly diminished in the EP2 (-/-) and EP4 (-/-) lines but not in the EP1 -/- or EP3 -/- lines. Because the actions of other hormonal systems that regulate blood pressure differ between **sexes**, we compared the roles of individual EP receptors in males and **females**. We found that the relative contribution of each EP-receptor subclass

was

strikingly different in males from that in **females**. In **females**, the EP2 and EP4 receptors, which signal by stimulating adenylate cyclase, mediate the major portion of the vasodepressor **response** to PGE2. In males, the EP2 receptor has a modest effect, but most of the vasodepressor effect is mediated by the phospholipase C-coupled EP1 receptor. Finally in male mice, the EP3 receptor actively opposes the vasodepressor actions of **PGE(2)**. Thus the hemodynamic actions of **PGE(2)** are mediated through complex interactions of several EP-receptor subtypes, and the role of individual EP receptors differs dramatically in males from that in **females**.

These differences may contribute to **sexual** dimorphism of blood pressure regulation.

L25 ANSWER 5 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:664971 SCISEARCH
THE GENUINE ARTICLE: 229BM
TITLE: Glial-neuronal interactions in the neuroendocrine control of mammalian puberty: Facilitatory effects of gonadal steroids
AUTHOR: Ojeda S R (Reprint); Ma Y J
CORPORATE SOURCE: OREGON HLTH SCI UNIV, DIV NEUROSCI, OREGON REG PRIMATE RES
RES CTR, 505 NW 185TH AVE, BEAVERTON, OR 97006 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF NEUROBIOLOGY, (15 SEP 1999) Vol. 40, No. 4, PP. 528-540.
PUBLISHER: JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0022-3034.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 95
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB It is now clear that astroglial cells actively contribute to both the generation and how of information within the central nervous system, In the hypothalamus, astrocytes regulate the secretory activity of neuroendocrine neurons, A small subset of these neurons secrete luteinizing hormone-releasing hormone (LHRH), a neuropeptide essential for **sexual** development and adult reproductive function, Astrocytes stimulate LHRH secretion via cell-cell signaling mechanisms involving growth factors recognized by receptors with either serine/threonine or tyrosine kinase activity. Two members of the epidermal growth factor (EGF) family and their respective tyrosine kinase receptors appear to play key roles in this regulatory process. Transforming growth factor-alpha (TGF alpha) and its distant congeners, the neuregulins (NRGs), are produced in hypothalamic astrocytes, They stimulate LHRH secretion indirectly, via activation of erbB-1/erbB-2 and erbB-4/erbB-2 receptor complexes also located on astrocytes, Activation of these receptors leads to release of prostaglandin E-2 (PGE(2)), which then binds to specific receptors on LHRH neurons to elicit LHRH secretion. Gonadal steroids facilitate this glia-to-neuron communication process by acting at three different steps along the signaling pathway, They (a) increase astrocytic gene expression of at least one of the EGF-related ligands (TGF alpha), (b) increase expression of at least two of the receptors (erbB-4 and erbB-2), and (c) enhance the LHRH **response** to PGE(2) by up-regulating in LHRH neurons the expression of specific PGE (2) receptor isoforms. Focal overexpression of TGF alpha in either the median eminence or preoptic area of the hypothalamus accelerates puberty, Conversely, blockade of either TGF alpha or NRG hypothalamic actions delays the process. Thus, both TGF alpha and NRGs appear to be physiological components of the central neuroendocrine mechanism controlling the initiation of **female** puberty. By facilitating growth factor signaling pathways in the hypothalamus, ovarian steroids accelerate the pace and progression of the pubertal process. (C) 1999

John

Wiley & Sons, Inc.

AB . . . secretory activity of neuroendocrine neurons, A small subset of these neurons secrete luteinizing hormone-releasing hormone (LHRH), a neuropeptide essential for **sexual** development and adult reproductive function, Astrocytes stimulate LHRH secretion via cell-cell signaling mechanisms involving growth factors recognized by receptors with. . . of erbB-1/erbB-2 and erbB-4/erbB-2 receptor complexes also located on astrocytes, Activation of these receptors leads to release of prostaglandin E-2 (PGE(2)), which then binds to specific receptors on LHRH neurons to elicit LHRH secretion. Gonadal steroids facilitate this glia-to-neuron communication process. . . (TGF alpha), (b) increase expression of at least two of the receptors (erbB-4 and erbB-2), and (c) enhance the LHRH **response** to PGE(2) by up-regulating in LHRH neurons the expression of specific PGE (2) receptor isoforms. Focal overexpression of TGF alpha in either the median eminence or preoptic area of the hypothalamus accelerates puberty,. . . Thus, both TGF alpha and NRGs appear to be physiological components of the central neuroendocrine mechanism controlling the initiation of **female** puberty. By facilitating growth factor signaling pathways in the hypothalamus, ovarian steroids accelerate the pace and progression of the pubertal. . .

=> d 125 6-10 ibib abs

L25 ANSWER 6 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:412549 SCISEARCH
THE GENUINE ARTICLE: 199MZ
TITLE: Cytokines inhibit sexual behavior in female rats: II.
Prostaglandins mediate the suppressive effects of
interleukin-1 beta
AUTHOR: Avitsur R (Reprint); Weidenfeld J; Yirmiya R
CORPORATE SOURCE: HEBREW UNIV JERUSALEM, DEPT PSYCHOL, MT SCOPUS, IL-91905
JERUSALEM, ISRAEL (Reprint); HADASSAH UNIV HOSP, DEPT
NEUROL, IL-91120 JERUSALEM, ISRAEL
COUNTRY OF AUTHOR: ISRAEL
SOURCE: BRAIN BEHAVIOR AND IMMUNITY, (MAR 1999) Vol. 13, No. 1,
pp. 33-45.
Publisher: ACADEMIC PRESS INC, 525 B ST, STE 1900, SAN
DIEGO, CA 92101-4495.
ISSN: 0889-1591.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 57

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The proinflammatory cytokine interleukin-1 (IL-1) induces several
behavioral alterations that are characteristic of illness, such as
anorexia and reduced locomotor and social activity. We have recently
demonstrated that IL-1 inhibits **sexual** activity, motivation and
attractivity in **female**, but not in male rats following either
central or peripheral administration. In the present study we examined
the involvement of prostaglandin (PG) synthesis in mediating IL-1-induced
suppression of **female sexual** behavior. Administration
of the cyclooxygenase blockers indomethacin or ibuprofen completely
prevented IL-1-induced suppression of **female sexual**
behavior, including the reduction in preceptive behavior, the lordosis
response to a male's mounts, and the preference for a
sexually active partner. In a subsequent study, ex-vivo release of
hypothalamic **PGE**(2) and the secretion of corticosterone (CS)
were measured in males and estrous **females** following IL-1
administration. At the same time and dose of IL-1 administration that
significantly reduced **sexual** behavior in **female** but
not male rats, IL-1 produced a significant increase in **PGE**(2)
release in **female**, but not in male rats. In contrast, IL-1
induced a significant elevation of serum CS levels in males but not in
females. These findings suggest that PG synthesis is involved in
mediating the effects of IL-1 on **female sexual**
behavior. Furthermore, differential secretion of PGs and CS may underlie
the gender difference in the effects of IL-1 on **sexual** behavior.
(C) 1999 Academic Press.

L25 ANSWER 7 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:219564 SCISEARCH
THE GENUINE ARTICLE: 175KM
TITLE: Seminal plasma components stimulate interleukin-8 and
interleukin-10 release
AUTHOR: Denison F C (Reprint); Grant V E; Calder A A; Kelly R W
CORPORATE SOURCE: UNIV EDINBURGH, DEPT OBSTET & GYNAECOL, CTR REPROD BIOL,
37 CHALMERS ST, EDINBURGH EH3 9ET, MIDLOTHIAN, SCOTLAND
(Reprint); UNIV EDINBURGH, MRC, CTR REPROD BIOL, REPROD
BIOL UNIT, EDINBURGH EH3 9ET, MIDLOTHIAN, SCOTLAND
COUNTRY OF AUTHOR: SCOTLAND
SOURCE: MOLECULAR HUMAN REPRODUCTION, (MAR 1999) Vol. 5, No. 3,

pp. 220-226.

Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD
OX2 6DP, ENGLAND.

ISSN: 1360-9947.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 40

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Human seminal plasma has potent anti-inflammatory properties which are thought to confer a survival advantage to the spermatozoa within the hostile **female genital** tract. In contrast, a profound pro-inflammatory leukocytosis has been observed post-coitus in animals and

humans. Whether components of seminal plasma are involved in initiating this leukocytic reaction is not known. This study investigated the effect of human seminal plasma, a seminal plasma fraction and its principal constituent prostaglandins, prostaglandin E-2 (**PGE**(2)) and 19-hydroxy **PGE**, on the release of the pro-inflammatory neutrophil chemotactic factor interleukin-8 (IL-8) and the anti-inflammatory cytokines interleukin-10 (IL-10) and secretory leukocyte

protease inhibitor (SLPI). The tissues studied were non-pregnant cervical explants, peripheral blood and the monocyte cell line U937. Seminal plasma

fraction (SPF) significantly ($P < 0.05$) stimulated release of IL-8 and inhibited release of SLPI from non-pregnant cervical explants. SPF, **PGE**2 and 19-hydroxy **PGE** significantly ($P < 0.005$) stimulated IL-8 release from peripheral blood and U937 cells. 19-hydroxy **PGE** was significantly ($P < 0.005$) more effective than **PGE**2 in stimulating IL-8 release. Seminal plasma, SPF and **PGE**, significantly ($P < 0.05$) stimulated IL-10 release from U937 cells. 19-hydroxy **PGE** stimulated IL-10 release from U937 cells but this failed to reach significance. Release of IL-10 by cervical explants and SLPI by

peripheral blood and U937 cells were below the detection limit of the assays employed. We suggest that the anti- and pro-inflammatory immune **responses** which seminal plasma induces might act in combination initially to promote sperm survival and then to facilitate their removal from the **female genital** tract.

L25 ANSWER 8 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 1998:939912 SCISEARCH

THE GENUINE ARTICLE: BL98W

TITLE: Vasopressin-induced antipyresis - Sex- and experience-dependent febrile responses

AUTHOR: Pittman Q J (Reprint); Chen X H; Mouihate A; Martin S
CORPORATE SOURCE: UNIV CALGARY, HLTH SCI CTR, DEPT PHYSIOL & BIOPHYS, FAC MED, 3330 HOSP DR NW, CALGARY, AB T2N 4N1, CANADA (Reprint); UNIV CALGARY, FAC MED, NEUROSCI RES GRP, CALGARY, AB T2N 4N1, CANADA; MT ST VINCENT UNIV, DEPT BIOL, HALIFAX, NS B3M 2J6, CANADA

COUNTRY OF AUTHOR: CANADA

SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (NOV 1998) Vol. 856, pp. 53-61.

Publisher: NEW YORK ACAD SCIENCES, 2 EAST 63RD ST, NEW YORK, NY 10021.

ISSN: 0077-8923.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE

LANGUAGE: English
REFERENCE COUNT: 45

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB There is now good evidence that vasopressin (AVP) acts, in the male rat, as a neurotransmitter in the ventral septal area to reduce fever. In light of the well known **sexual** dimorphism in the AVP innervation of the brain, we asked if **female** rats would (a) display fevers different from those seen in male rats, (b) **respond** to AVP with antipyresis, (c) display evidence of endogenous AVP-induced antipyresis during fever, and (d) display altered fevers and AVP involvement as a function of hormonal status. Our experiments indicate that **female** rats display larger fevers to intracranial prostaglandin E-2 (PGE(2)) but not to systemic lipopolysaccharide or interleukin-1 beta than do male rats. The larger fevers may be due, in part, to a lack of AVP-induced antipyresis, as an AVP antagonist elevates PGE(2) fever in male but not in **female** rats and dialysates of the ventral septal area show increased AVP levels only in male rats during defervescence. Nonetheless, **females respond** to exogenous AVP with antipyresis. Throughout late pregnancy, parturition, and lactation, PGE(2) fevers are reduced, but this appears to be due to a general suppression of autonomic output not involving enhanced AVP antipyresis. Fevers due to lipopolysaccharide and interleukin-1 beta are also suppressed at this time, and in some animals, fevers are dramatically suppressed at about the time of parturition. Our results indicate that **female** rats may utilize different strategies for antipyresis than do male rats and that hormonal status may influence both peripherally generated and centrally activated fevers.

L25 ANSWER 9 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1998:113935 SCISEARCH
THE GENUINE ARTICLE: BK30S

TITLE: Aromatase expression in health and disease
AUTHOR: Simpson E R (Reprint); Zhao Y; Agarwal V R; Michael M D; Bulun S E; Hinshelwood M M; Graham-Lorence S; Sun T J; Fisher C R; Qin K N; Mendelson C R
CORPORATE SOURCE: UNIV TEXAS, SW MED CTR, CECIL H & IDA GREEN CTR REPROD BIOL SCI, DALLAS, TX 75235 (Reprint); UNIV TEXAS, SW MED CTR, DEPT OBSTET GYNECOL, DALLAS, TX 75235; UNIV TEXAS, SW MED CTR, DEPT BIOCHEM, DALLAS, TX 75235
COUNTRY OF AUTHOR: USA
SOURCE: RECENT PROGRESS IN HORMONE RESEARCH, (JAN 1997) Vol. 52, pp. 185-214.
Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4410.
ISSN: 0079-9963.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 123

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Family 19 of the P450 superfamily is **responsible** for the conversion of C-19 androgenic steroids to the corresponding estrogens, a reaction known as aromatization, since it involves conversion of the Delta(4)-3-one A-ring of the androgens to the corresponding phenolic A-ring characteristic of estrogens. Its members occur throughout the entire vertebrate phylum. The reaction mechanism of aromatase is very interesting from a chemical point of view and has been studied

extensively; however, a detailed examination of structure-function relationships has not been possible due to lack of a crystal structure. Recent attempts to model the three-dimensional structure of aromatase have permitted a model that accounts for the reaction mechanism and predicts the location of aromatase inhibitors. The gene encoding human aromatase has been cloned and characterized and shown to be unusual compared to genes encoding other P450 enzymes, since there are a number of untranslated first exons that occur in aromatase transcripts in a tissue-specific fashion, due to differential splicing as a consequence of the use of tissue-specific promoters. Thus, expression in ovary utilizes a proximal promoter that is regulated primarily by cAMP. On the other hand, expression in placenta utilizes a distal promoter that is located at least 40 kb upstream of the start of transcription and that is regulated by retinoids. Other promoters are employed in brain and adipose tissue. In the latter case, class 1 cytokines such as IL-6 and IL-11 as well as TNF alpha are important regulatory factors. PGE(2) is also an important regulator of aromatase expression in adipose mesenchymal cells via cAMP and PGE(2) appears to be a major factor produced by breast tumors that stimulates estrogen biosynthesis in local mesenchymal sites. In all of the splicing events involved in the use of these various promoters, a common 3'-splice junction is employed that is located upstream of the start of translation; thus, the coding regions of the transcripts-and hence the protein-are identical regardless of the tissue site of expression; what differ in a tissue-specific fashion are the 5'-ends of the transcripts. This pattern of expression has great significance both from a phylogenetic and ontogenetic standpoint as well as for the physiology and pathophysiology of estrogen formation. Recently, a number of mutations of the aromatase gene have been described, which give rise to complete estrogen deficiency. In **females** this results in virilization in-utero and primary amenorrhea with hypergonadotropic hypogonadism at the time of puberty. In men the most striking feature is continued linear bone growth beyond the time of puberty, delayed bone age, and failure of epiphyseal closure, thus indicating an important role of estrogens in bone metabolism in men. In both **sexes** the symptoms can be alleviated by estrogen administration.

L25 ANSWER 10 OF 69 SCISEARCH COPYRIGHT 2001 ISI (R)
 ACCESSION NUMBER: 97:868473 SCISEARCH
 THE GENUINE ARTICLE: YG185
 TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women
 AUTHOR: Farker K (Reprint); Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth H W; Hoffmann A; Oettel M
 CORPORATE SOURCE: UNIV JENA, INST CLIN PHARMACOL, D-07740 JENA, GERMANY (Reprint); UNIV MARBURG, DEPT PEDIAT, D-35033 MARBURG, GERMANY; UNIV JENA, INST MED STAT INFORMAT & DOCUMENTAT, D-07740 JENA, GERMANY; JENAPHARM GMBH, D-07745 JENA, GERMANY
 COUNTRY OF AUTHOR: GERMANY
 SOURCE: PROSTAGLANDINS, (SEP 1997) Vol. 54, No. 3, pp. 655-664. Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010. ISSN: 0090-6980.
 DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 18

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E-2 (**PGE** (2)), 6-keto-PGF(1 alpha), thromboxane B-2 (TxB(2)) and their metabolites **PGE-M** (11 alpha-hydroxy-9,15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanic acid), 2,3-dinor-6-keto-PGF(1 alpha), 2,3-dinor-TxB(2) and 11-dehydro-TxB(2) were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young nonpregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, **PGE**(2) and TxB(2) were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and post-menopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

=> s 125 range=,1998

'CN' IS NOT A VALID FIELD CODE

'CN' IS NOT A VALID FIELD CODE

4 FILES SEARCHED...

'CN' IS NOT A VALID FIELD CODE

5 FILES SEARCHED...

'CN' IS NOT A VALID FIELD CODE

8 FILES SEARCHED...

12 FILES SEARCHED...

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH

FIELD CODE - 'AND' OPERATOR ASSUMED 'L65 (S) L66'

L29 36 L25

=> d ibib abs 1-10

L29 ANSWER 1 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 1998:939912 SCISEARCH

THE GENUINE ARTICLE: BL98W

TITLE: Vasopressin-induced antipyresis - Sex- and experience-dependent febrile responses

AUTHOR: Pittman Q J (Reprint); Chen X H; Mouihate A; Martin S

CORPORATE SOURCE: UNIV CALGARY, HLTH SCI CTR, DEPT PHYSIOL & BIOPHYS, FAC MED, 3330 HOSP DR NW, CALGARY, AB T2N 4N1, CANADA (Reprint); UNIV CALGARY, FAC MED, NEUROSCI RES GRP, CALGARY, AB T2N 4N1, CANADA; MT ST VINCENT UNIV, DEPT BIOL, HALIFAX, NS B3M 2J6, CANADA

COUNTRY OF AUTHOR: CANADA

SOURCE: ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (NOV 1998) Vol. 856, pp. 53-61.

Publisher: NEW YORK ACAD SCIENCES, 2 EAST 63RD ST, NEW
YORK, NY 10021.
ISSN: 0077-8923.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 45

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB There is now good evidence that vasopressin (AVP) acts, in the male rat, as a neurotransmitter in the ventral septal area to reduce fever. In light of the well known **sexual** dimorphism in the AVP innervation of the brain, we asked if **female** rats would (a) display fevers different from those seen in male rats, (b) **respond** to AVP with antipyresis, (c) display evidence of endogenous AVP-induced antipyresis during fever, and (d) display altered fevers and AVP involvement as a function of hormonal status. Our experiments indicate that **female** rats display larger fevers to intracranial prostaglandin E-2 (**PGE**(2)) but not to systemic lipopolysaccharide or interleukin-1 beta than do male rats. The larger fevers may be due, in part, to a lack of AVP-induced antipyresis, as an AVP antagonist elevates **PGE**(2) fever in male but not in **female** rats and dialysates of the ventral septal area show increased AVP levels only in male rats during defervescence. Nonetheless, **females respond** to exogenous AVP with antipyresis. Throughout late pregnancy, parturition, and lactation, **PGE**(2) fevers are reduced, but this appears to be due to a general suppression of autonomic output not involving enhanced AVP antipyresis. Fevers due to lipopolysaccharide and interleukin-1 beta are also suppressed at this time, and in some animals, fevers are dramatically suppressed at about the time of parturition. Our results indicate that **female** rats may utilize different strategies for antipyresis than do male rats and that hormonal status may influence both peripherally generated and centrally activated fevers.

L29 ANSWER 2 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 1998:113935 SCISEARCH

THE GENUINE ARTICLE: BK30S

TITLE: Aromatase expression in health and disease

AUTHOR: Simpson E R (Reprint); Zhao Y; Agarwal V R; Michael M D; Bulun S E; Hinshelwood M M; Graham-Lorence S; Sun T J; Fisher C R; Qin K N; Mendelson C R

CORPORATE SOURCE: UNIV TEXAS, SW MED CTR, CECIL H & IDA GREEN CTR REPROD BIOL SCI, DALLAS, TX 75235 (Reprint); UNIV TEXAS, SW MED CTR, DEPT OBSTET GYNECOL, DALLAS, TX 75235; UNIV TEXAS,

SW

MED CTR, DEPT BIOCHEM, DALLAS, TX 75235

COUNTRY OF AUTHOR: USA

SOURCE: RECENT PROGRESS IN HORMONE RESEARCH, (JAN 1997) Vol. 52, pp. 185-214.

Publisher: ENDOCRINE SOC, 4350 EAST WEST HIGHWAY SUITE 500, BETHESDA, MD 20814-4410.

ISSN: 0079-9963.

DOCUMENT TYPE: General Review; Journal

LANGUAGE: English

REFERENCE COUNT: 123

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Family 19 of the P450 superfamily is **responsible** for the conversion of C-19 androgenic steroids to the corresponding estrogens, a

reaction known as aromatization, since it involves conversion of the Delta(4)-3-one A-ring of the androgens to the corresponding phenolic A-ring characteristic of estrogens. Its members occur throughout the entire vertebrate phylum. The reaction mechanism of aromatase is very interesting from a chemical point of view and has been studied extensively; however, a detailed examination of structure-function relationships has not been possible due to lack of a crystal structure. Recent attempts to model the three-dimensional structure of aromatase

have

permitted a model that accounts for the reaction mechanism and predicts the location of aromatase inhibitors. The gene encoding human aromatase has been cloned and characterized and shown to be unusual compared to genes encoding other P450 enzymes, since there are a number of untranslated first exons that occur in aromatase transcripts in a tissue-specific fashion, due to differential splicing as a consequence of the use of tissue-specific promoters. Thus, expression in ovary utilizes

a

proximal promoter that is regulated primarily by cAMP. On the other hand, expression in placenta utilizes a distal promoter that is located at

least

40 kb upstream of the start of transcription and that is regulated by retinoids. Other promoters are employed in brain and adipose tissue. In the latter case, class 1 cytokines such as IL-6 and IL-11 as well as TNF alpha are important regulatory factors. **PGE**(2) is also an important regulator of aromatase expression in adipose mesenchymal cells via cAMP and **PGE**(2) appears to be a major factor produced by breast tumors that stimulates estrogen biosynthesis in local mesenchymal sites. In all of the splicing events involved in the use of these various promoters, a common 3'-splice junction is employed that is located upstream of the start of translation; thus, the coding regions of the transcripts-and hence the protein-are identical regardless of the tissue site of expression; what differ in a tissue-specific fashion are the 5'-ends of the transcripts. This pattern of expression has great significance both from a phylogenetic and ontogenetic standpoint as well as for the physiology and pathophysiology of estrogen formation.

Recently,

a number of mutations of the aromatase gene have been described, which give rise to complete estrogen deficiency. In **females** this results in virilization in utero and primary amenorrhea with hypergonadotropic hypogonadism at the time of puberty. In men the most striking feature is continued linear bone growth beyond the time of puberty, delayed bone age, and failure of epiphyseal closure, thus indicating an important role of estrogens in bone metabolism in men. In both **sexes** the symptoms can be alleviated by estrogen administration.

L29 ANSWER 3 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 97:868473 SCISEARCH

THE GENUINE ARTICLE: YG185

TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women

AUTHOR: Farker K (Reprint); Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth H W; Hoffmann A; Oettel M

CORPORATE SOURCE: UNIV JENA, INST CLIN PHARMACOL, D-07740 JENA, GERMANY (Reprint); UNIV MARBURG, DEPT PEDIAT, D-35033 MARBURG, GERMANY; UNIV JENA, INST MED STAT INFORMAT & DOCUMENTAT, D-07740 JENA, GERMANY; JENAPHARM GMBH, D-07745 JENA, GERMANY

COUNTRY OF AUTHOR: GERMANY

SOURCE: PROSTAGLANDINS, (SEP 1997) Vol. 54, No. 3, pp. 655-664.
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE
AMERICAS, NEW YORK, NY 10010.
ISSN: 0090-6980.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 18

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E-2 (PGE (2)), 6-keto-PGF(1 alpha), thromboxane B-2 (TxB(2)) and their metabolites PGE-M (11 alpha-hydroxy-9,15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanic acid), 2,3-dinor-6-keto-PGF(1 alpha), 2,3-dinor-TxB(2) and 11-dehydro-TxB(2) were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young nonpregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, PGE(2) and TxB(2) were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and post-menopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L29 ANSWER 4 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 97:647831 SCISEARCH

THE GENUINE ARTICLE: XT349

TITLE: Enhancement by sex hormones of the osteoregulatory effects

of mechanical loading and prostaglandins in explants of rat ulnae

AUTHOR: Cheng M Z; Zaman G; Rawlinson S C F; Pitsillides A A; Suswillo R F L; Lanyon L E (Reprint)

CORPORATE SOURCE: UNIV LONDON ROYAL VET COLL, ROYAL COLL ST, LONDON NW1 0TU,

ENGLAND (Reprint); UNIV LONDON ROYAL VET COLL, LONDON NW1 0TU, ENGLAND

COUNTRY OF AUTHOR: ENGLAND

SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (SEP 1997) Vol. 12, No. 9, pp. 1424-1430.
Publisher: BLACKWELL SCIENCE INC, 350 MAIN ST, MALDEN, MA 02148.
ISSN: 0884-0431.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 41

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Explants of ulnae from 5-week-old male and **female** rats were

cleaned of marrow and soft tissue and, in the presence and absence of 10(-8) M 17 beta-estradiol (E2) or 5 alpha-dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 mu epsilon, 1 Hz, 8 minutes, maximum strain rate 25,000 mu epsilon/s), prostaglandin E-2 (PGE(2)) and prostacyclin (PGI(2)) (10(-6) M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from males and **females**, as indicated by incorporation of [H-3]thymidine into DNA and [H-3]proline into collagen. E2 and DHT both increased [H-3]thymidine and [H-3]proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **response** in [H-3] thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and PGE(2) or PGI(2). The interaction between loading and E-2 for [H-3]proline incorporation was not mimicked by these prostanoids. In males the synergism in [H-3]proline incorporation seen between loading and DHT was mimicked by that between PGI(2) and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**. Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are reproduced in some but not all cases by E2 and prostaglandins. E2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.

L29 ANSWER 5 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
 ACCESSION NUMBER: 97:387605 SCISEARCH
 THE GENUINE ARTICLE: WY547
 TITLE: A cytokine switch induced by human seminal plasma: An immune modulation with implications for sexually transmitted disease
 AUTHOR: Kelly R W (Reprint); Carr G G; Critchley H O D
 CORPORATE SOURCE: UNIV EDINBURGH, CTR REPROD BIOL, MRC, REPROD BIOL UNIT, 37 CHALMERS ST, EDINBURGH EH3 9EW, MIDLOTHIAN, SCOTLAND (Reprint); UNIV EDINBURGH, CTR REPROD BIOL, DEPT OBSTET & GYNAECOL, EDINBURGH EH3 9EW, MIDLOTHIAN, SCOTLAND
 COUNTRY OF AUTHOR: SCOTLAND
 SOURCE: HUMAN REPRODUCTION, (APR 1997) Vol. 12, No. 4, pp. 677-681
 Publisher: OXFORD UNIV PRESS, WALTON ST JOURNALS DEPT, OXFORD, ENGLAND OX2 6DP.
 ISSN: 0268-1161.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE; CLIN
 LANGUAGE: English
 REFERENCE COUNT: 36

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The immunosuppressive activity of human seminal plasma may be one factor in the aetiology of **sexually** transmitted disease and could be particularly important for the spread of human immunodeficiency

virus (HIV), The advent of virus that can preferentially infect Langerhans cells of the **genital** mucosa underscores the relevance of seminal plasma effects, Virally infected cells are eradicated by the killing activity of T cells and natural killer (NK) cells and this cytotoxicity is stimulated by IL-12 (previously known as natural killer cell stimulatory factor) and partly inhibited by IL-10 (previously known as cytokine synthesis inhibitory factor), We have examined the effects of human seminal plasma on the production of these key cytokines, Cytokine production was measured in rapidly diluted, fresh, lipopolysaccharide (LPS)-stimulated, whole blood since this provided leukocytes with minimal exposure to prostaglandin, Prostaglandin concentrations and cytokine release were measured by ELISA, Addition of human seminal plasma diluted up to 100 000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio ($P < 0.02$), A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma), This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (PGE) and 19-OH PGE (the main prostaglandin constituent of human seminal plasma), Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution, However, its activity could be restored by the addition of synthetic PGE and 19-hydroxy PGE, A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower **female**-reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells, In addition, the stimulation of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells, The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower **genital** tract, with ablation of cell-mediated **responses** and immunosurveillance. (C) European Society for Human Reproduction and Embryology.

L29 ANSWER 6 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
 ACCESSION NUMBER: 97:386214 SCISEARCH
 THE GENUINE ARTICLE: WY626
 TITLE: Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE(2) fever
 AUTHOR: Chen X (Reprint); Landgraf R; Pittman Q J
 CORPORATE SOURCE: UNIV CALGARY, DEPT PHYSIOL & BIOPHYS, NEUROSCI RES GRP, 3330 HOSP DR NW, CALGARY, AB T2N 4N1, CANADA (Reprint); MAX PLANCK INST PSYCHIAT, INST CLIN, D-80804 MUNICH, GERMANY
 COUNTRY OF AUTHOR: CANADA; GERMANY
 SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-REGULATORY INTEGRATIVE AND COMPARATIVE PHYSIOLOGY, (MAY 1997) Vol. 41, No. 5, pp. R1664-R1669.
 Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
 ISSN: 0363-6119.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: English
 REFERENCE COUNT: 30

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less

well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses**. We therefore examined the temperature **responses** of conscious and urethan-anesthetized rats of both **sexes** to centrally administered prostaglandin E-2 (**PGE**(2)) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious [25 ng/5 μ l **PGE**(2) intracerebroventricularly (icv)] and anesthetized (VSA microdialyzed, 50 ng/5 μ l **PGE**(2) icv) **female** rats had higher fevers than did males. Infusion of an AVP V-1a receptor antagonist [1 nmol [d(CH₂)(5)Tyr(Me)]AVP] plus **PGE**(2) gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to **PGE**(2) in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in **PGE**(2) fever, which may be accounted for by the differential AVP release in the VSA.

L29 ANSWER 7 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 96:916161 SCISEARCH

THE GENUINE ARTICLE: VW830

TITLE: Self-reported sexual function and sexual arousability in women with epilepsy

AUTHOR: Morrell M J (Reprint); Guldner G T

CORPORATE SOURCE: STANFORD UNIV, MED CTR, DEPT NEUROL & NEUROL SCI, SCH MED,

STANFORD COMPREHENS EPILEPSY CTR, H3160, STANFORD, CA 94307 (Reprint)

COUNTRY OF AUTHOR: USA

SOURCE: EPILEPSIA, (DEC 1996) Vol. 37, No. 12, pp. 1204-1210. Publisher: LIPPINCOTT-RAVEN PUBL, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106. ISSN: 0013-9580.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 35

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Purpose: Women with epilepsy are at risk for sexual dysfunction but the

frequency and types of dysfunction have not been well characterized.

Methods: Self-reported sexual function was evaluated in 116 women aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17

with primary generalized epilepsy (PGE). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure. Standardized inventories assessed sexual functioning, sexual arousability and anxiety, sexual behavior, and depression.

Results: Although **sexual** experience was not reduced, **women** with **PGE** and **LRE** reported significantly less **sexual** arousability and **women** with **LRE** reported significantly more **sexual** anxiety. **Women** with **LRE** experienced significantly more dyspareunia, vaginismus, **arousal** insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset.

Conclusions: In contrast to subjects of previous research, the women in

our study did not have a disorder of sexual desire, but more than one third experienced disorders of sexual arousal, implying a physiological deficit. Although the etiology for these arousal phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed in the treatment of sexual disorders.

L29 ANSWER 8 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 96:479396 SCISEARCH

THE GENUINE ARTICLE: UT138

TITLE: PROGESTERONE AND DEXAMETHASONE STIMULATE PROLIFERATION AND

DIFFERENTIATION OF OSTEOPROGENITORS AND PROGENITORS FOR ADIPOCYTES AND MACROPHAGES IN CELL-POPULATIONS DERIVED FROM ADULT-RAT VERTEBRAE

AUTHOR: ISHIDA Y (Reprint); TERTINEGG I; HEERSCHKE J N M

CORPORATE SOURCE: UNIV TORONTO, FAC DENT, ROOM 400, 124 EDWARD ST, TORONTO, ON M5G 1G6, CANADA (Reprint)

COUNTRY OF AUTHOR: CANADA

SOURCE: JOURNAL OF BONE AND MINERAL RESEARCH, (JUL 1996) Vol. 11, No. 7, pp. 921-930.
ISSN: 0884-0431.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 36

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in a-minimum essential medium plus 10% fetal calf serum (standard

medium) and then subculturing the outgrowth cells in standard medium plus 50 μ g/ml of ascorbic acid, 5 mM beta-glycerophosphate, and with or without Frog or Dex. On day 6 of culture, these populations were analyzed for CAMP **responses** to parathyroid hormone (PTH), prostaglandin E(2) (**PGE**(2)), and isoproterenol (IPT). Increases in intracellular CAMP were seen in **response** to PTH, **PGE**(2), and IPT, and culturing in medium containing Frog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Frog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Frog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Frog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of alpha-naphthyl butyrate esterase (alpha-NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED2-positive colonies (macrophages). Frog-treated cultures contained more colonies of small spindle-shaped alpha-NBE-positive cells and fewer colonies of small round alpha-NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Frog and Dex. The data also suggest that the effects of frog and Dex differ qualitatively and quantitatively.

L29 ANSWER 9 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
 ACCESSION NUMBER: 95:80195 SCISEARCH
 THE GENUINE ARTICLE: QB499
 TITLE: MESENTERIC VASCULAR-RESPONSES TO VASOPRESSIN DURING
 DEVELOPMENT OF DOCA-SALT HYPERTENSION IN MALE AND FEMALE
 RATS
 AUTHOR: STALLONE J N (Reprint)
 CORPORATE SOURCE: NE OHIO UNIV, COLL MED, DEPT PHYSIOL, 4209 STATE RT 44,
 POB 95, ROOTSTOWN, OH, 44272 (Reprint)
 COUNTRY OF AUTHOR: USA
 SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-REGULATORY INTEGRATIVE AND
 COMPARATIVE PHYSIOLOGY, (JAN 1995) Vol. 37, No. 1, pp.
 R40-R49.
 ISSN: 0363-6119.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: ENGLISH
 REFERENCE COUNT: 58

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Deoxycorticosterone acetate (DOCA)-salt hypertension develops to a greater extent in male (M) than in **female** (F) rats. To determine the role of the vasculature, reactivity to arginine vasopressin (AVP) and prostanoid output were examined in the isolated perfused mesenteric vasculature of hypertensive (HT) and normotensive-control (NTC) M and F rats after acute (1-wk) and chronic (4-wk) DOCA-salt treatment. Systolic blood pressure was significantly higher in M than in F HT rats (187 +/- 3 vs. 151 +/- 3 mmHg after 4 wk; $P < 0.02$). After acute treatment, vascular reactivity to AVP (maximal perfusion pressure) in HT was elevated in M (181 +/- 18 mmHg; $P < 0.02$) but not in F (135 +/- 6 mmHg) compared with NTC (90 +/- 6 mmHg, M vs. 119 +/- 5 mmHg, F). After chronic treatment, vascular reactivity to AVP in HT was elevated in both **sexes** ($P < 0.02$), although more in F (175 +/- 13 mmHg) than in M (141 +/- 11 mmHg). In contrast, vascular **responsiveness** to phenylephrine did not differ significantly between M and F NTC or HT preparations after either acute or chronic treatment. **Sex** differences in basal and AVP-induced 6-ketoprostaglandin (6-keto-PG) F-1 alpha and **PGE**(2) output by HT and NTC vasculature were reciprocal to **sex** differences in the vasoconstriction **responses** to AVP. After acute treatment, AVP-stimulated 6-keto-PGF(1 alpha) output by HT was elevated slightly in F (33.6 +/- 1.7 ng/3 min; P less than or equal to 0.02) but not in M (49.9 +/- 4.3 ng/3 min) compared with NTC (23.5 +/- 2.6 ng/3 min, F vs. 34.7 +/- 4.9 ng/3 min, M). After chronic treatment, output by HT was enhanced in both **sexes** (P less than or equal to 0.02), although more in M (109 +/- 15.4 ng/3 min) than in F (68 +/- 6.6 ng/3 min). These findings suggest that **sex** differences in the relative balance between AVP-induced vasoconstriction and vasodilatory prostanoid release may contribute to male-**female** differences in mesenteric vascular reactivity to AVP in NT and that disturbances in this balance may be **responsible**, at least in part, for the **sex**- and time-dependent changes in reactivity to AVP observed during the development of DOCA-salt hypertension.

L29 ANSWER 10 OF 36 SCISEARCH COPYRIGHT 2001 ISI (R)
 ACCESSION NUMBER: 94:151690 SCISEARCH
 THE GENUINE ARTICLE: MY446
 TITLE: GENDER-RELATED RESPONSE TO A TERT-BUTYL
 HYDROPEROXIDE-INDUCED OXIDATION IN HUMAN NEONATAL TISSUE

AUTHOR: LAVOIE J C; CHESSEX P (Reprint)
 CORPORATE SOURCE: UNIV MONTREAL, HOP STE JUSTINE, DEPT PEDIAT, RES CTR, 3175
 CHEMIN COTE ST CATHERINE, MONTREAL H3T 1C5, PQ, CANADA (Reprint); UNIV MONTREAL, HOP STE JUSTINE, DEPT PEDIAT, RES CTR, MONTREAL H3T 1C5, PQ, CANADA; UNIV MONTREAL, HOP STE JUSTINE, DEPT PEDIAT, PERINATAL SERV, MONTREAL H3T 1C5, PQ, CANADA
 COUNTRY OF AUTHOR: CANADA
 SOURCE: FREE RADICAL BIOLOGY AND MEDICINE, (MAR 1994) Vol. 16, No. 3, pp. 307-313.
 ISSN: 0891-5849.
 DOCUMENT TYPE: Article; Journal
 FILE SEGMENT: LIFE
 LANGUAGE: ENGLISH
 REFERENCE COUNT: 31

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Reports of gender-related differences in the activity of enzymes involved in the metabolism of intracellular antioxidants, led us to verify whether the prostaglandin **response** to tert-butyl hydroperoxide (TBH) differed according to the **sex** of infants. Segments of human umbilical veins were perfused in the presence or absence of TBH (0.25 mmol/l, and 1.0 mmol/l). Because TBH is quenched in the cell by glutathione peroxidase, total glutathione concentrations and the production of glutathione-dependent prostaglandins (**PGE**(2) and **PCF2** alpha) as well as membrane-derived eicosanoids (**PGI**(2) and thromboxane) were measured in the eluate. In veins from boys, TBH induced a sustained **response** for glutathione only, which was increased ($p < 0.05$). In **female**-derived tissue, the hydroperoxide induced a different **response** according to the dose of TBH. At 0.25 mmol/l, a drop ($p < 0.005$) in **PGF**(2) alpha was associated with a rise ($p < 0.001$) in thromboxane. At 1.0 mmol/l, TBH had an opposite effect-there was a rise ($p < 0.01$) in **PGE**(2) and **PGI**(2). The prostaglandin concentrations were not proportional to the oxidative stimulus, suggesting a critical level of TBH at which the oxidative state differs in tissues derived from boys or **girls**.

=> d ibib abs 11-20

L29 ANSWER 11 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
 ACCESSION NUMBER: 1997-0518264 PASCAL
 COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
 TITLE (IN ENGLISH): Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae
 AUTHOR: MING ZHAO CHENG; ZAMAN G.; RAWLINSON S. C. F.; PITSILLIDES A. A.; SUSWILLO R. F. L.; LANYON L. E.
 CORPORATE SOURCE: The Royal Veterinary College, University of London, London, United Kingdom
 SOURCE: Journal of bone and mineral research, (1997), 12(9), 1424-1430, 42 refs.
 ISSN: 0884-0431 CODEN: JBMREJ
 DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic
 COUNTRY: United States
 LANGUAGE: English
 AVAILABILITY: INIST-21114, 354000069214160120
 AN 1997-0518264 PASCAL
 CP Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
 AB Explants of ulnae from 5-week-old male and **female** rats were cleaned of marrow and soft tissue and, in the presence and absence of 10.sup.-.sup.8 M 17.beta.-estradiol (E2) or 5.alpha.-dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 .mu..epsilon., 1 Hz, 8 minutes, maximum strain rate 25,000 .mu..epsilon./s), prostaglandin E.sub.2 (PGE.sub.2) and prostacyclin (PGI.sub.2) (10.sup.-.sup.6 M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from males and **females**, as indicated by incorporation of [.sup.3H]thymidine into DNA and [.sup.3H]proline into collagen. E2 and DHT both increased [.sup.3H]thymidine and [.sup.3H]proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **response** in [.sup.3H]thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and PGE.sub.2 or PGI.sub.2. The interaction between loading and E2 for [.sup.3H]proline incorporation was not mimicked by these prostanoids. In males the synergism in [.sup.3H]proline incorporation seen between loading and DHT was mimicked by that between PGI.sub.2 and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**.

 Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are reproduced in some but not all cases by E2 and prostaglandins. E2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.
 L29 ANSWER 12 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
 ACCESSION NUMBER: 1997-0326904 PASCAL
 COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
 TITLE (IN ENGLISH): Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE.sub.2 fever
 AUTHOR: CHEN X.; LANDGRAF R.; PITTMAN Q. J.
 CORPORATE SOURCE: Neuroscience Research Group, Department of Physiology and Biophysics, University of Calgary, Calgary, Alberta, T2N 4N1, Canada; Max Planck Institute of Psychiatry, Clinical Institute, 80804 Munich, Germany,
 SOURCE: Federal Republic of American journal of physiology. Regulatory, integrative and comparative physiology, (1997), 41(5),
 R1664-R1669, 30 refs.
 ISSN: 0363-6119 CODEN: AJPRDO
 DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670E, 354000065609740390
AN 1997-0326904 PASCAL
CP Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses**. We therefore examined the temperature **responses** of conscious and urethan-anesthetized rats of both **sexes** to centrally administered prostaglandin E2 (PGE2) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious [25 ng/5 .mu.l PGE .sub.2 intracerebroventricularly (icv)] and anesthetized (VSA microdialyzed, 50 ng/5 .mu.l PGE2 icv) **female** rats had higher fevers than did males. Infusion of an AVP V1a receptor antagonist [1 nmol [d(CH2.sub.2).sub.5Tyr(Me)]AVP] plus PGE.sub.2 gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to PGE.sub.2 in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in PGE2 fever, which may be accounted for by the differential AVP release in the VSA.

L29 ANSWER 13 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.

ACCESSION NUMBER: 1997-0307382 PASCAL

COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.

TITLE (IN ENGLISH): A cytokine switch induced by human seminal plasma :
an

immune modulation with implications for sexually transmitted disease

AUTHOR: KELLY R. W.; CARR G. G.; CRITCHLEY H. O. D.

CORPORATE SOURCE: Medical Research Council Reproductive Biology Unit,
University of Edinburgh Centre for Reproductive
Biology, 37 Chalmers Street, Edinburgh EH3 9EW,

United

Kingdom; Department of Obstetrics and Gynaecology,
University of Edinburgh Centre for Reproductive
Biology, 37 Chalmers Street, Edinburgh EH3 9EW,

United

Kingdom

SOURCE: Human reproduction : (Oxford), (1997), 12(4),
677-681,

36 refs.

ISSN: 0268-1161 CODEN: HUREEE

DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: United Kingdom

LANGUAGE: English

AVAILABILITY: INIST-21337, 354000065485320100

AN 1997-0307382 PASCAL

CP Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.

AB The immunosuppressive activity of human seminal plasma may be one factor in the aetiology of **sexually** transmitted disease and could be particularly important for the spread of human immunodeficiency virus (HIV). The advent of virus that can preferentially infect Langerhans cells of the **genital** mucosa underscores the relevance of

seminal plasma effects. Virally infected cells are eradicated by the killing activity of T cells and natural killer (NK) cells and this cytotoxicity is stimulated by IL-12 (previously known as natural killer cell stimulatory factor) and partly inhibited by IL-10 (previously known as cytokine synthesis inhibitory factor). We have examined the effects of human seminal plasma on the production of these key cytokines. Cytokine production was measured in rapidly diluted, fresh, lipopolysaccharide (LPS)-stimulated, whole blood since this provided leukocytes with minimal exposure to prostaglandin. Prostaglandin concentrations and cytokine release were measured by ELISA. Addition of human seminal plasma diluted up to 100 000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio ($P < 0.02$). A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma). This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (PGE) and 19-OH PGE (the main prostaglandin constituent of human seminal plasma). Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution. However, its activity could be restored by the addition of synthetic PGE and 19-hydroxy PGE. A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower female reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells. In addition, the stimulation of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells. The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower genital tract, with ablation of cell-mediated responses and immunosurveillance.

L29 ANSWER 14 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
 ACCESSION NUMBER: 1997-0131769 PASCAL
 COPYRIGHT NOTICE: Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
 TITLE (IN ENGLISH): Self-reported sexual function and sexual arousability in women with epilepsy
 AUTHOR: MORRELL M. J.; GULDNER G. T.
 CORPORATE SOURCE: Department of Neurology and Neurological Sciences, Stanford Comprehensive Epilepsy Center, Stanford University School of Medicine, Stanford, California, United States
 SOURCE: Epilepsia : (Copenhagen), (1996), 37(12), 1204-1210, 39 refs.
 ISSN: 0013-9580 CODEN: EPILAK
 DOCUMENT TYPE: Journal
 BIBLIOGRAPHIC LEVEL: Analytic
 COUNTRY: United States
 LANGUAGE: English
 AVAILABILITY: INIST-1145, 354000061163140090
 AN 1997-0131769 PASCAL
 CP Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
 AB Purpose : Women with epilepsy are at risk for sexual dysfunction but the frequency and types of dysfunction have not been well characterized. Methods : Self-reported sexual function was

evaluated in 116 **women** aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17 with primary generalized epilepsy (**PGE**). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure.

Standardized

inventories assessed **sexual** functioning, **sexual** arousability and anxiety, **sexual** behavior, and depression. Results.- Although **sexual** experience was not reduced, **women** with **PGE** and LRE reported significantly less **sexual** arousability and **women** with LRE reported significantly more **sexual** anxiety. **Women** with LRE experienced significantly more dyspareunia, vaginismus, **arousal** insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset. Conclusions : In contrast to subjects of previous research, the **women** in our study did not have a disorder of **sexual desire**, but more than one third experienced disorders of **sexual arousal**, implying a physiological deficit. Although the etiology for these **arousal** phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed

in

the treatment of **sexual** disorders.

L29 ANSWER 15 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.
ACCESSION NUMBER: 1996-0374537 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1996 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Progesterone and dexamethasone stimulate proliferation
and differentiation of osteoprogenitors and progenitors for adipocytes and macrophages in cell populations derived from adult rat vertebrae
AUTHOR: ISHIDA Y.; TERTINEGG I.; HEERSCHKE J. N. M.
CORPORATE SOURCE: Faculty of Dentistry, University of Toronto, Toronto Ontario, Canada
SOURCE: Journal of bone and mineral research, (1996), 11(7), 921-930, 36 refs.
ISSN: 0884-0431 CODEN: JBMREJ
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-21114, 354000060117470070
AN 1996-0374537 PASCAL
CP Copyright .COPYRGT. 1996 INIST-CNRS. All rights reserved.
AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in .alpha.-minimum essential medium plus 10% fetal calf serum (standard medium) and then subculturing the outgrowth cells in standard medium plus 50 .mu.g/ml of ascorbic acid, 5 mM .beta.-glycerophosphate, and with or without Prog or Dex. On day 6 of culture, these populations were analyzed for cAMP **responses** to parathyroid hormone (PTH),

prostaglandin E.sub.2 (PGE.sub.2), and isoproterenol (IPT). Increases in intracellular cAMP were seen in **response** to PTH, PGH.sub.2, and IPT, and culturing in medium containing Prog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Prog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Prog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Prog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of .alpha.-naphthyl butyrate esterase (.alpha.-NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED2-positive colonies (macrophages). Prog-treated cultures contained more colonies of small spindle-shaped .alpha.-NBE-positive cells and fewer colonies of small round .alpha.-NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Prog and Dex. The data also suggest that the effects of Prog and Dex differ qualitatively and quantitatively.

L29 ANSWER 16 OF 36 PASCAL COPYRIGHT 2001 INIST-CNRS. ALL RIGHTS RESERVED.

ACCESSION NUMBER: 1995-0168851 PASCAL

COPYRIGHT NOTICE: Copyright .COPYRGT. 1995 INIST-CNRS. All rights reserved.

TITLE (IN ENGLISH): Mesenteric vascular responses to vasopressin during development of DOCA-salt hypertension in male and female rats

AUTHOR: STALLONE J. N.

CORPORATE SOURCE: Northeastern Ohio univ. coll. medicine, dep. physiology, Rootstown OH 44272, United States

SOURCE: American journal of physiology. Regulatory, integrative and comparative physiology, (1995),

37(1),

R40-R49, 58 refs.

ISSN: 0363-6119 CODEN: AJPRDO

DOCUMENT TYPE: Journal

BIBLIOGRAPHIC LEVEL: Analytic

COUNTRY: United States

LANGUAGE: English

AVAILABILITY: INIST-670E, 354000059782010070

AN 1995-0168851 PASCAL

CP Copyright .COPYRGT. 1995 INIST-CNRS. All rights reserved.

AB Deoxycorticosterone acetate (DOCA)-salt hypertension develops to a greater extent in male (M) than in **female** (F) rats. To determine the role of the vasculature, reactivity to arginine vasopressin

(AVP) and prostanoid output were examined in the isolated perfused mesenteric vasculature of hypertensive (HT) and normotensive-control (NTC) M and F rats after acute (1-wk) and chronic (4-wk) DOCA-salt treatment. Systolic blood pressure was significantly higher in M than in F HT rats (187. \pm .3 vs. 151. \pm .3 mmHg after 4 wk; $P < 0.02$). After acute treatment, vascular reactivity to AVP (maximal perfusion pressure) in HT was elevated in M (181. \pm .18 mmHg; $P < 0.02$) but not in F (135. \pm .6 mmHg) compared with NTC (90. \pm .6 mmHg, M vs. 119. \pm .5 mmHg, F). After chronic treatment, vascular reactivity to AVP in HT was elevated in both **sexes** ($P < 0.02$), although more in F (175. \pm .13 mmHg) than in M

(141. \pm .11 mmHg). In contrast, vascular **responsiveness** to phenylephrine did not differ significantly between M and F NTC or HT preparations after either acute or chronic treatment. **Sex** differences in basal and AVP-induced 6-ketoprostaglandin (6-keto-PG) F.sub.1.sub..alpha. and PGE.sub.2 output by HT and NTC vasculature were reciprocal to **sex** differences in the vasoconstriction **responses** to AVP. After acute treatment, AVP-stimulated 6-keto-PGF.sub.1.sub..alpha. output by HT was elevated slightly in F (33.6. \pm .1.7 ng/3 min; P \leq 0.02) but not in M (49.9. \pm .4.3 ng/3 min) compared with NTC (23.5. \pm .2.6 ng/3 min, F vs. 34.7. \pm .4.9 ng/3

min, M). After chronic treatment, output by HT was enhanced in both **sexes** (P \leq 0.02), although more in M (109. \pm .15.4 ng/3 min) than in F (68. \pm .6.6 ng/3 min). These findings suggest that **sex** differences in the relative balance between AVP-induced vasoconstriction and vasodilatory prostanoid release may contribute to male-female differences in mesenteric vascular reactivity to AVP in NT and that disturbances in this balance may be **responsible**, at least in part, for the **sex**- and time-dependent changes in reactivity to AVP observed during the development of DOCA-salt hypertension

L29 ANSWER 17 OF 36 TOXLIT

ACCESSION NUMBER: 1998:15340 TOXLIT

DOCUMENT NUMBER: CA-128-030641P

TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women.

AUTHOR: Farker K; Schweer H; Vollandt R; Nassr N; Nagel U; Seyberth

CORPORATE SOURCE: HW; Hoffmann A; Oettel M
Institute of Clinical Pharmacology, Friedrich Schiller University, Jena, Jena

SOURCE: Prostaglandins, (1997). Vol. 54, No. 3, pp. 655-664.
CODEN: PRGLBA. ISSN. 0090-6980.

PUB. COUNTRY:-- -- GERMANY, FEDERAL REPUBLIC OF

DOCUMENT TYPE: Journal; Journal Article

FILE SEGMENT: CA

LANGUAGE: English

OTHER SOURCE: CA 128:30641

ENTRY MONTH: 199804

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E2 (PGE2), 6-keto-PGF1.alpha., thromboxane B2 (TxB2) and their metabolites PGE-M (11.alpha.-hydroxy-9,15-dioxo-2,3,4,5,20-pentanor-19-carboxyprostanic acid), 2,3-dinor-6-keto-PGF1.alpha., 2,3-dinor-TxB2 and 11-dehydro-TxB2 were detd. by gas chromatog.-triple stage quadrupole mass spectrometry (GC/MS/MS) in 41 young non-pregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, PGE2 and TxB2 were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and postmenopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins.

Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L29 ANSWER 18 OF 36 TOXLIT

ACCESSION NUMBER: 1988:75313 TOXLIT

DOCUMENT NUMBER: CA-109-067612P

TITLE: Effects of female sex hormones and pregnancy on gallbladder

prostaglandin synthesis.

AUTHOR: Hoover EL; Jaffe BM; Webb H; England DW

CORPORATE SOURCE: Health Sci. Cent., State Univ. New York, Brooklyn

SOURCE: Arch. Surg. (Chicago), (1988). Vol. 123, No. 6, pp. 705-8.

CODEN: ARSUAX. ISSN. 0004-0010.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

FILE SEGMENT: CA

LANGUAGE: English

OTHER SOURCE: CA 109:67612

ENTRY MONTH: 198810

AB To investigate whether **female sex** hormones and pregnancy induced increased gallbladder synthesis of PGI₂ and prostaglandin E (**PGE**), an in vitro incubation chamber was used to quantitate the effects of progesterone, estrogen, pregnancy, and pregnancy plus a 2%-cholesterol diet on mucosal and serosal PGI₂ and **PGE** prodn. by the rabbit gallbladder. Neither the **female sex** hormones nor pregnancy alone caused an increase in PGI₂ or **PGE** synthesis. The gallbladders of cholesterol-fed, pregnant rabbits demonstrated increases only in serosal synthesis of PGI₂. This increased prodn. was equiv. to that noted for gallbladders from nonpregnant rabbits fed a high-cholesterol diet. There were no increases in mucosal synthesis of **PGE** or of PGI₂. Thus, neither elevated levels of progesterone or estrogen nor pregnancy is directly **responsible** for the increased PGI₂ activity in the **female** gallbladder; conversely, this effect seems to be mediated by the increased biliary concns. of cholesterol.

L29 ANSWER 19 OF 36 TOXLIT

ACCESSION NUMBER: 1982:84100 TOXLIT

DOCUMENT NUMBER: CA-097-139356S

TITLE: Prostaglandin-mediated inhibition of lymphokine secretion in normal individuals and patients with progressive systemic sclerosis (scleroderma, PSS).

AUTHOR: Kelly RH; Miller DH; Rodnan GP; Hagmann J

CORPORATE SOURCE: Sch. Med., Univ. Pittsburgh, Pittsburgh

SOURCE: Agents Actions, (1982). Vol. 12, No. 4, pp. 471-7.

CODEN: AGACBH. ISSN. 0065-4299.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

FILE SEGMENT: CA

LANGUAGE: English

OTHER SOURCE: CA 97:139356

ENTRY MONTH: 198211

AB The sensitivity of peripheral blood lymphocytes to E-type prostaglandin (**PGE**)-mediated inhibition of lymphokine secretion was examd. in 3 groups of individuals; normal controls, hospitalized patients, and

patients with progressive systemic sclerosis. Leukocytes were stimulated by a polyclonal T-cell activator, phytohemagglutinin, and the release of the lymphokine, leukocyte migration inhibitory factor (LIF), was measured in the presence or absence of exogenous PGE₂ (I) [363-24-6] using a direct agarose droplet migration inhibition technique. Leukocytes of scleroderma patients were hyporesponsive to **PGE** (i.e., lymphokine secretion by these cells was not inhibited at concns. of PGE₂ of 2.8 .times. 10⁻⁸-2.8 .times. 10⁻⁵M). In addn., a marked **sex** difference in **PGE responsiveness** existed among normal controls, whereby **females** were hyporesponsive during the latter half of the menstrual cycle. This deficit may facilitate, in part, the development of connective tissue diseases in **women** of child-bearing age. The inability to suppress lymphokine prodn. and arrest persistent immune reactivity, coupled with the known ability of lymphokines to augment fibroblast collagen prodn., offers a reasonable explanation for the accumulation of tissue collagen in scleroderma.

L29 ANSWER 20 OF 36 TOXLIT

ACCESSION NUMBER: 1981:40993 TOXLIT

DOCUMENT NUMBER: CA-095-018389E

TITLE: Effect of adrenoceptor blockers and MAOI on postcoital utero-oviductal contractility, sperm transport, and sperm attachment to eggs in rabbits.

AUTHOR: Marsafy YM; Hafez ES E

CORPORATE SOURCE: Sch. Med., Wayne State Univ., Detroit

SOURCE: Arch. Androl, (1981). Vol. 6, No. 4, pp. 307-16.

CODEN: ARANDR.

FILE SEGMENT: CA

LANGUAGE: English

OTHER SOURCE: CA 95:18389

ENTRY MONTH: 198108

AB Adult **female** rabbits were injected with phenoxybenzamine (alpha-adrenoceptor blocker), propranolol (beta-adrenoceptor blocker), and

isocarboxazid (monoamine oxidase inhibitor, MAOI). (59-96-1 Phenoxybenzamine)(525-66-6 Propranolol)(59-63-2 Isocarboxazid) Physiol. saline was injected in the controls. Treatment started 1 day before mating and continued until autopsy. At 3, 12, and 24 h postcoitum (PC), utero-oviductal contractions were recorded in vivo using micro-balloon-ended, fluid-filled catheters as pressure receptors. At 24 h PC, all groups were autopsied and flushings of the contralateral oviducts were evaluated for both sperm count and no. of sperm attached and/or penetrating the eggs. Phenoxybenzamine induced redn. in the amplitude and frequency of both active contractions and resting pressure fluctuations. This suppressive effect was more remarkable at 12 and 24 h PC than at 3 h PC. Propranolol induced incoordination and instability in the uterooviductal resting pressure and increased the amplitude of

isthmus

contraction, and in frequency of uterine and ampullary contractions.

Such

excitatory effects were more pronounced at 3 h PC than at 12 and 24 h PC. More effective alpha-adrenergic blockade seemed to coincide with postovulatory progesterone dominance, increase in concn., and **response to genital tract PGE**. More effective beta-adrenoceptor blockade coincided with preovulatory estrogen dominance and increased concn. and **response to genital tissue PGF**. At 24 h PC, sperm count was reduced in the oviduct and fewer nos.

of

sperm were attached to the eggs in phenoxybenzamine- and

propranolol-treated **females**. Phenoxybenzamine-induced suppression of utero-oviductal contractions facilitated oviductal sperm ascent. Propranolol caused irregular sperm transport with rapid loss in the peritoneal cavity. At 24 h PC, oviductal sperm count increased and an excessive no. of sperm were attached to the eggs in isocarboxazid-treated rabbits. The pharmacol. of sperm transport is discussed in relation to infertility and contraception.

=>

=> d 21-30 ibib abs

L29 ANSWER 21 OF 36 TOXLIT

ACCESSION NUMBER: 1981:31132 TOXLIT

DOCUMENT NUMBER: CA-094-167790K

TITLE: Effects of cannabinoids and female exposure on the pituitary-testicular axis in mice: possible involvement of prostaglandins.

AUTHOR: Dalterio S; Bartke A; Harper MJ K; Huffman R; Sweeney C
CORPORATE SOURCE: Health Sci. Cent., Univ. Texas, San Antonio
SOURCE: Biol. Reprod, (1981). Vol. 24, No. 2, pp. 315-22.
CODEN: BIREBV.

FILE SEGMENT: CA

LANGUAGE: English

OTHER SOURCE: CA 94:167790

ENTRY MONTH: 198106

AB In immature (30-35-day-old) mice, a single dose of DELTA9-THC(I), the main

psychoactive constituent of marihuana, decreased plasma testosterone (T), LH, and FSH levels, but the same dose of a nonpsychoactive component, cannabinol (CBN), had not effect. (1972-08-3 DELTA.9-Tetrahydrocannabinol) (58-22-0 Testosterone) (9002-67-9 LH) (9002-68-0 FSH) (521-35-7 Cannabinol) Chronic exposure to THC, CBN, or cannabidiol (CBD), beginning at 30 days of age through adulthood, influenced the endocrine **responses** to a **sexually** receptive **female**. (13956-29-1 Cannabidiol) Thus, wts. of testes and seminal vesicles were reduced in males from all cannabinoid-treated groups

on the day after exposure to a **female**, compared with treated males housed in all-male groups. Plasma FSH concns. were elevated in CBN-exposed mice, regardless of social experience, while plasma T levels were increased after an encounter with a **female** in all but THC-treated males. Plasma LH levels and testicular **responsiveness** to gonadotropins in vitro were reduced in THC- and CBN-treated mice exposed to a **female**. In contrast, in THC- or CBN-treated males maintained in all-male groups, T prodn. in vitro was significantly elevated. Alterations in prostaglandin (PG) concns. may mediate these effects of cannabinoids and **sexual** encounter since prodn. of PG in vitro by testis and pituitary was reduced by exposing cannabinoid-treated males to **female**-related stimuli. In contrast, **sexual** encounter increased PGF, but had no effect on **PGE** prodn. by pituitary or testes obtained from oil-treated controls. Both psychoactive and nonpsychoactive constituents of marihuana

are capable of altering the function of the pituitary-gonadal axis and of influencing the endocrine **responsivity** to **female**-related exteroceptive cues in male mice.

L29 ANSWER 22 OF 36 Elsevier BIOBASE COPYRIGHT 2001 Elsevier Science B.V.
 ACCESSION NUMBER: 1997256480 Elsevier BIOBASE
 TITLE: Measurements of urinary prostaglandins in young ovulatory women during the menstrual cycle and in postmenopausal women
 AUTHOR: Farker K.; Schweer H.; Vollandt R.; Nassr N.; Nagel U.; Seyberth H.W.; Hoffmann A.; Oettel M.
 CORPORATE SOURCE: Dr. K. Farker, Institute of Clinical Pharmacology, Friedrich-Schiller-University Jena, D-07740 Jena, Germany.
 SOURCE: Prostaglandins, (1997), 54/3 (655-664), 18 reference(s)
 CODEN: PRGLBA ISSN: 0090-6980
 PUBLISHER ITEM IDENT.: S0090698097001317
 DOCUMENT TYPE: Journal; Article
 COUNTRY: United States
 LANGUAGE: English
 SUMMARY LANGUAGE: English

AB The purpose of the present work was to study the prostaglandin excretion in young nonpregnant ovulatory **women** during the menstrual cycle on the one hand and in postmenopausal **women** on the other hand and to investigate the influence of **female sex** hormones (estradiol, progesterone) on urinary prostanoid excretion. Urinary excretion rates of prostaglandin E.sub.2 (**PGE**.sub.2), 6-keto-PGF.sub.1.alpha., thromboxane B.sub.2 (**TXB**.sub.2) and their metabolites **PGE**-M (11.alpha.- hydroxy-9,15-dioxo-2,3,4,5,20-pentanoic-19-carboxyprostanic acid), 2,3- dinor-6-keto-PGF.sub.1.alpha., 2,3-dinor-**TXB**.sub.2 and 11-dehydro-**TXB**.sub.2 were determined by gas chromatography-triple stage quadrupole mass spectrometry (GC/MS/MS) in
 41 young non pregnant **women** during the follicular phase and during the luteal phase and in 23 postmenopausal **women**. Excretion rates of all urinary prostanoids were not significantly different in the follicular phase when compared with the luteal phase. In contrast to the young ovulatory **women**, **PGE**.sub.2 and **TXB**.sub.2 were significantly higher in postmenopausal **women**. Concerning the other prostaglandins significant differences between these groups of **women** did not exist. Although serum levels of estradiol and progesterone were different in young and postmenopausal **women**, **sex** hormones have not been shown to correlate with prostaglandins. Our data do not suggest **sex** hormones to be **responsible** for the difference in the prostaglandin excretion in **women** of reproductive age and in **women** in the menopause. Further systematic investigations into age dependency of prostaglandin excretion in **women** are necessary.

L29 ANSWER 23 OF 36 Elsevier BIOBASE COPYRIGHT 2001 Elsevier Science B.V.
 ACCESSION NUMBER: 1997133358 Elsevier BIOBASE
 TITLE: Differential ventral septal vasopressin release is associated with sexual dimorphism in PGE.sub.2 fever
 AUTHOR: Chen X.; Landgraf R.; Pittman Q.J.
 CORPORATE SOURCE: X. Chen, Neuroscience Research Group, Dept. of Physiology and Biophysics, University of Calgary,
 3330 Hospital Dr. NW, Calgary, Alta. T2N 4N1, Canada.
 SOURCE: American Journal of Physiology - Regulatory Integrative and Comparative Physiology, (1997), 272/5 41-5 (R1664-R1669), 30 reference(s)
 CODEN: AJPRDO ISSN: 0363-6119

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The vasopressinergic innervation of the ventral septal area (VSA) has been shown to be implicated in antipyresis. Because this system is less well developed in **female** rats, we hypothesized that **female** rats would display exaggerated febrile **responses**. We therefore examined the temperature **responses** of conscious and urethane-anesthetized rats of both **sexes** to centrally administered prostaglandin E.sub.2 (**PGE**.sub.2) and correlated these **responses** with the release and action of endogenous arginine vasopressin (AVP) in the VSA. Both conscious .cents.25 ng/5 .mu.l **PGE**.sub.2 intracerebroventricularly (icv)! and anesthetized (VSA microdialyzed, 50 ng/5 .mu.l **PGE**.sub.2 icv) **female** rats had higher fevers than did males. Infusion of an AVP V(1a) receptor antagonist .cents.1 nmol! d(CH.sub.2).sub.5Tyr(Me)!AVP! plus **PGE**.sub.2 gave rise to higher fevers in males but not in **females**. Measurements of AVP in microdialysates of the VSA showed that the release of endogenous AVP was increased in **response** to **PGE**.sub.2 in males only. Baseline AVP release in both **sexes** was similar. The results suggest that there is a **sex**-related difference in **PGE**.sub.2 fever, which may be accounted for by the differential AVP release in the VSA.

L29 ANSWER 24 OF 36 Elsevier BIOBASE COPYRIGHT 2001 Elsevier Science B.V.
ACCESSION NUMBER: 1996190112 Elsevier BIOBASE
TITLE: Self-reported sexual function and sexual arousability in women with epilepsy
AUTHOR: Morrell M.J.; Guldner G.T.
CORPORATE SOURCE: Dr. M.J. Morrell, Dept. of Neurology/Neurological Sci., Stanford University Medical Center, Stanford, CA
94307, United States.

SOURCE: Epilepsia, (1996), 37/12 (1204-1210)
CODEN: EPILAK ISSN: 0013-9580

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Purpose: **Women** with epilepsy are at risk for **sexual** dysfunction but the frequency and types of dysfunction have not been well

characterized. Methods: Self-reported **sexual** function was evaluated in 116 **women** aged 18-65 years with epilepsy and no concomitant medical or psychiatric illness, including 99 with localization-related epilepsy (LRE) and 17 with primary generalized epilepsy (**PGE**). Variables evaluated included seizure frequency, age of seizure onset, and antiepileptic drug (AED) exposure.

Standardized inventories assessed **sexual** functioning, **sexual** arousability and anxiety, **sexual** behavior, and depression. Results: Although **sexual** experience was not reduced, **women** with **PGE** and LRE reported significantly less **sexual** arousability and **women** with LRE reported significantly more **sexual** anxiety. **Women** with LRE experienced significantly more dyspareunia, vaginismus, arousal insufficiency, and **sexual** dissatisfaction, whereas **women** with **PGE** experienced anorgasmia and **sexual** dissatisfaction. **Sexual** symptoms were not

associated with seizure frequency, AED exposure, **sexual** experience, depression, or prepubertal seizure onset. Conclusions: In contrast to subjects of previous research, the **women** in our study did not have a disorder of **sexual desire**, but more than one third experienced disorders of **sexual arousal**, implying a physiological deficit. Although the etiology for these **arousal** phase dysfunctions has not been defined, such conditions are treatable and warrant referral to a gynecologist versed in the treatment of **sexual** disorders.

L29 ANSWER 25 OF 36 LIFESCI COPYRIGHT 2001 CSA
ACCESSION NUMBER: 1998:33681 LIFESCI
TITLE: Enhancement by sex hormones of the osteoregulatory effects of mechanical loading and prostaglandins in explants of rat ulnae
AUTHOR: Cheng, Ming Zhao; Zaman, G.; Rawlinson, S.C.F.; Pitsillides, A.A.; Suswillo, R.F.L.; Lanyon, L.E.*
CORPORATE SOURCE: Royal Veterinary College, University of London, Royal College Street, London NW1 0TU, UK
SOURCE: J. BONE MINER. RES., (19970900) vol. 12, no. 9, pp. 1424-1430.
ISSN: 0884-0431.
DOCUMENT TYPE: Journal
FILE SEGMENT: T
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Explants of ulnae from 5-week-old male and **female** rats were cleaned of marrow and soft tissue and, in the presence and absence of 10 super(-8) M 17 beta -estradiol (E2) or 5 alpha -dihydrotestosterone (DHT), mechanically loaded or treated with exogenous prostanoids previously shown to be produced during loading. Over an 18-h period, mechanical loading (peak strain 1300 mu epsilon, 1 Hz, 8 minutes, maximum strain rate 25,000 mu epsilon /s), prostaglandin E sub(2) (PGE sub(2)) and prostacyclin (PGI sub(2)) (10 super(-6) M), each separately produced quantitatively similar increases in cell proliferation and matrix production in bones from males and **females**, as indicated by incorporation of [super(3)H]thymidine into DNA and [super(3)H]proline into collagen. E2 and DHT both increased [super(3)H]thymidine and [super(3)H]proline incorporations, E2 producing greater increases in **females** than in males. Indomethacin abrogated the effects of loading, but had no effects on those of **sex** hormones. Loading, or prostanoids, together with **sex** hormones, produced **responses** generally equal to or greater than the addition of the individual influences acting independently. In **females** there was a synergistic **response** in [super(3)H]thymidine incorporation between loading and E2, which was quantitatively similar to the interaction between E2 and PGE sub(2) or PGI sub(2). The interaction between loading and E2 for [super(3)H]proline incorporation was not mimicked by these prostanoids. In males the synergism in [super(3)H]proline incorporation seen between loading and DHT was mimicked by that between PGI sub(2) and DHT. We conclude that loading stimulates increased bone cell proliferation and matrix production in situ through a prostanoid-dependent mechanism. This **response** is equal in size in males and **females**. Estrogen and testosterone increase proliferation and matrix production through a mechanism independent of prostanoid production. The interactions between loading and hormones are

reproduced in some but not all cases by E2 and prostaglandins. E2 with loading and prostaglandins has greater effects in **female** bones, while DHT with loading and prostaglandins has greater effects in males.

L29 ANSWER 26 OF 36 LIFESCI COPYRIGHT 2001 CSA

ACCESSION NUMBER: 97:14091 LIFESCI

TITLE: Progesterone and dexamethasone stimulate proliferation and differentiation of osteoprogenitors and progenitors for adipocytes and macrophages in cell populations derived

from

adult rat vertebrae

AUTHOR: Ishida, Y.; Tertinegg, I.; Heersche, J.N.M.

CORPORATE SOURCE: Faculty of Dentistry, University of Toronto, Room 400, 124 Edward Street, Toronto, Ontario M5G 1G6, Canada

SOURCE: J. BONE MINER. RES., (1996) vol. 11, no. 7, pp. 921-930. ISSN: 0884-0431.

DOCUMENT TYPE: Journal

FILE SEGMENT: T

LANGUAGE: English

SUMMARY LANGUAGE: English

AB We investigated the effects of the **sex** hormone progesterone (Prog) and the synthetic glucocorticoid dexamethasone (Dex) on proliferation and differentiation of progenitor cells of osteogenic, adipocytic, and hemopoietic lineages in cell populations derived from explants of adult **female** rat lumbar vertebrae. The cell populations were obtained by culturing bone explants in plasma clots immersed in alpha -minimum essential medium plus 10% fetal calf serum (standard medium) and then subculturing the outgrowth cells in standard medium plus 50 mu g/ml of ascorbic acid, 5 mM beta -glycerophosphate, and with or without Prog or Dex. On day 6 of culture, these populations were analyzed for cAMP **responses** to parathyroid hormone (PTH), prostaglandin E sub(2) (PGE sub(2)), and isoproterenol (IPT). Increases in intracellular cAMP were seen in **response** to PTH, PGE sub(2), and IPT, and culturing in medium containing Prog increased these **responses**. At various time periods between days 4-27 of culture, the cultures were evaluated for the presence of bone nodules, alkaline phosphatase (AP)-positive colonies, adipocytes, monocytes, and macrophages. Prog and Dex increased the number of bone nodules and AP-positive colonies. The effect of Prog on bone nodule formation was smaller than that of Dex. In addition, the effect of Dex on bone nodule formation was evident after 10 days of culture, while the Prog-induced effects became significant at days 16-20 of culture. Both hormones also increased the number of Sudan IV-positive colonies (adipocytes), certain types of alpha -naphthyl butyrate esterase (alpha -NBE)-positive colonies (monocytes, macrophages, and T-lymphocytes), and ED2-positive colonies (macrophages). Prog-treated cultures contained more colonies of small spindle-shaped alpha -NBE-positive cells and fewer colonies of small round alpha -NBE-positive cells when compared with Dex-treated cultures. These data indicate that cell populations derived from adult rat lumbar vertebrae contain, among others, osteoprogenitors and progenitors for adipocytes and macrophages that are stimulated to proliferate and differentiate by Prog and Dex. The data also suggest that the effects of Prog and Dex differ qualitatively and quantitatively.

L29 ANSWER 27 OF 36 LIFESCI COPYRIGHT 2001 CSA

ACCESSION NUMBER: 81:28933 LIFESCI

TITLE: Effect of Specific Estrogens on Prostaglandin Synthesis in Aorta and Thrombocytes of Female Pigeons.

AUTHOR: Subbiah, M.T.R.; Deitemeyer, D.; Yunker, R.; Gallon, L.

CORPORATE SOURCE: Dept. Med. Pathol., Lipid Res. Ctr., Univ. Cincinnati Med.

SOURCE: Ctr., Cincinnati, OH 45267, USA
PROC. SOC. EXP. BIOL. MED., (1981) vol. 166, no. 2, pp.
300-304.

DOCUMENT TYPE: Journal

FILE SEGMENT: X

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The effect of two major natural estrogens (estrone and 17 beta
-estradiol)

on prostaglandin biosynthesis from (super(14)C)arachidonic acid in
thrombocytes and aorta of **female** pigeons was compared with that
of a male **sex** hormone (testosterone). In the aorta, 17 beta
-estradiol stimulated the synthesis of 6-keto, PGF sub(1 alpha) and PGF
sub(2 alpha) but markedly reduced the synthesis of **PGE** sub(2).
Estrone on the other hand stimulated the synthesis of **PGE**
sub(2). Testosterone stimulated the synthesis of all prostaglandins in
the
aorta. In the thrombocytes, 17 beta -estradiol decreased aggregatory
response to arachidonic acid and synthesis of thromboxane B
sub(2). Estrone on the other hand increased aggregatory **response**
to arachidonic acid. Testosterone decreased the synthesis of thromboxane
B
sub(2).

L29 ANSWER 28 OF 36 CANCERLIT

ACCESSION NUMBER: 97302992 CANCERLIT

DOCUMENT NUMBER: 97302992

TITLE: A cytokine switch induced by human seminal plasma: an
immune modulation with implications for sexually
transmitted disease.

AUTHOR: Kelly R W; Carr G G; Critchley H O

CORPORATE SOURCE: Medical Research Council Reproductive Biology Unit,
University of Edinburgh Centre for Reproductive Biology,

UK

SOURCE: HUMAN REPRODUCTION, (1997). Vol. 12, No. 4, pp. 677-81.
Journal code: HRP. ISSN: 0268-1161.

DOCUMENT TYPE: (CLINICAL TRIAL)

Journal; Article; (JOURNAL ARTICLE)

FILE SEGMENT: MEDL; L; Priority Journals

LANGUAGE: English

OTHER SOURCE: MEDLINE 97302992

ENTRY MONTH: 199708

AB The immunosuppressive activity of human seminal plasma may be one factor
in the aetiology of **sexually** transmitted disease and could be
particularly important for the spread of human immunodeficiency virus
(HIV). The advent of virus that can preferentially infect Langerhans
cells

of the **genital** mucosa underscores the relevance of seminal
plasma effects. Virally infected cells are eradicated by the killing
activity of T cells and natural killer (NK) cells and this cytotoxicity
is

stimulated by IL-12 (previously known as natural killer cell stimulatory
factor) and partly inhibited by IL-10 (previously known as cytokine
synthesis inhibitory factor). We have examined the effects of human
seminal plasma on the production of these key cytokines. Cytokine
production was measured in rapidly diluted, fresh, lipopolysaccharide
(LPS)-stimulated, whole blood since this provided leukocytes with minimal
exposure to prostaglandin. Prostaglandin concentrations and cytokine
release were measured by ELISA. Addition of human seminal plasma diluted

up to 100,000 times (0.001%) to blood cell cultures led to a marked increase in the IL-10/IL-12 ratio ($P < 0.02$). A dose-dependent increase in the ratio was observed in five separate experiments, from a control value of 1 (no seminal plasma) to a mean value of 80 (1% seminal plasma). This cytokine switch was also seen when seminal plasma was substituted by pure prostaglandin E (PGE) and 19-OH PGE (the main prostaglandin constituent of human seminal plasma). Lipid-extracted seminal plasma was considerably less active at high dilutions than whole seminal plasma at the same dilution. However, its activity could be restored by the addition of synthetic PGE and 19-hydroxy PGE. A stimulation of IL-10 and a decrease in IL-12 in host-defence cells of the lower **female** reproductive tract will seriously affect the ability of cytotoxic T cells and NK cells to recognise and destroy virally infected cells. In addition, the stimulation of IL-10 will inhibit the release of the anti-HIV activity from CD8+ve cells. The cytokine switch reported here, activated by semen deposition, would exercise a key inhibitory control over vital immune defences in the lower **genital** tract, with ablation of cell-mediated **responses** and immunosurveillance.

L29 ANSWER 29 OF 36 CANCERLIT

ACCESSION NUMBER: 83097004 CANCERLIT

DOCUMENT NUMBER: 83097004

TITLE: Prostaglandin-mediated inhibition of lymphokine secretion in normal individuals and patients with progressive systemic sclerosis (scleroderma, PSS).

AUTHOR: Kelly R H; Miller D H; Rodnan G P; Hagmann J

CONTRACT NUMBER: FR-00056

SOURCE: AGENTS AND ACTIONS, (1982). Vol. 12, No. 4, pp. 471-7. Journal code: 2XZ. ISSN: 0065-4299.

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

FILE SEGMENT: MEDL; L; Priority Journals

LANGUAGE: English

OTHER SOURCE: MEDLINE 83097004

ENTRY MONTH: 198303

AB The sensitivity of peripheral blood lymphocytes to E-type prostaglandin-mediated inhibition of lymphokine secretion was examined in 3 groups of individuals; normal controls, hospitalized patients, and patients with progressive systemic sclerosis (PSS, scleroderma). Leukocytes were stimulated by a polyclonal T-cell activator, phytohemagglutinin, and the release of the lymphokine, leukocyte migration

inhibitory factor (LIF), was measured in the presence or absence of exogenous PGE₂ using a direct agarose droplet migration inhibition technique. Leukocytes of scleroderma patients were found to be hyporesponsive to E-type prostaglandin (i.e., lymphokine secretion by these cells was not inhibited at concentrations of PGE₂ between 2.8×10^{-8} and 2.8×10^{-5} M). In addition, a marked **sex** difference in **PGE responsiveness** was found to exist among normal controls, whereby **females** were hyporesponsive during the latter half of the menstrual cycle. It is possible that this deficit may facilitate, in part, the development of connective tissue diseases in **women** of childbearing age. The inability to suppress lymphokine production and arrest persistent immune reactivity, coupled with the known

ability of lymphokines to augment fibroblast collagen production, offers

a reasonable explanation for the accumulation of tissue collagen in scleroderma.

L29 ANSWER 30 OF 36 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD

ACCESSION NUMBER: 1998-15025 DRUGU T E

TITLE: Treatment of endometrial adenomatosis with tamoxifen in patients of reproductive age.

AUTHOR: Smetnik V P; Chernukha G E; Kushlinskii N E

CORPORATE SOURCE: Russian-Acad.Med.Sci.

LOCATION: Moscow, Russia

SOURCE: Byull.Eksp.Biol.Med. (125, No. 1, 93-97, 1998) 2 Tab. 18 Ref.

CODEN: BEBMAE ISSN: 0365-9615

AVAIL. OF DOC.: Scientific Center for Obstetrics, Gynecology and Perinatology, Russian Academy of Medical Sciences, Moscow, Russia.

LANGUAGE: English

DOCUMENT TYPE: Journal

FIELD AVAIL.: AB; LA; CT

FILE SEGMENT: Literature

AN 1998-15025 DRUGU T E

AB Courses of tamoxifen (TA) were effective against recurrent endometrial hyperplasia and adenomatosis in most of 51 women of reproductive age who had previously failed to respond to hormone therapy. Initial studies showed that most patients had reductions

in serum sex hormone binding globulin (SHBG), resulting in increased free blood estrogen levels; endometrial PGE levels were also elevated. In patients with complete morphological responses to TA, these changes were reversed and estrogen-17-beta (ES) receptors disappeared from the endometrium. However, in patients with partial or no responses, changes were smaller or absent, and in some cases total ES increased. While all women were initially infertile, pregnancy was established in 2 following TA treatment. TA produced no side-effects.

ABEX Methods 51 Women (mean age 30.9 yr) with recurrent endometrial hyperplasia and adenomatosis received TA (30 mg/day) for 3 mth, extended to 6 mth when incomplete responses were obtained. Controls were 11 healthy women (mean age 28.6 yr). Results Baseline studies showed that 76.6% of patients were overweight, 70.5% were hirsute, all had chronic anovulation, and 29.4% had cystic ovaries. Gonadotrophic hormones were essentially normal, though the LH/FSH ratio was elevated

in 29.4%. Total serum ES was normal but, because of reductions in SHBG in 86.3% of cases, free ES levels were elevated nearly 2-fold. ES

receptors were present in higher concentrations than in controls. Thus, patients were considered to have hyperestrogenemia resulting in hyperplasia. TA was followed by amenorrhea in 47.1% and regular or rare menstruation in 52.0%. Among the former and latter groups, TA eliminated adenomatosis

in 100% and 85.2% and eliminated all signs of hyperplasia in 62.5% and 41%; 1 patient in each group became pregnant. After extension of treatment

to 6 mth in patients with partial or no responses, only 3 remained with focal hyperplasia and 1 with focal adenomatosis requiring surgery. SHBG levels and free ES levels normalized in patients with good morphological responses but not in non-responders. (VH)

=> fil stng

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

	ENTRY	SESSION
FULL ESTIMATED COST	133.51	233.43

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
CA SUBSCRIBER PRICE	ENTRY	SESSION
	0.00	-0.59

FILE 'STNGUIDE' ENTERED AT 12:49:45 ON 14 MAY 2001
 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT
 COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY, JAPAN SCIENCE
 AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
 LAST RELOADED: May 11, 2001 (20010511/UP).

=> FIL F2, F6-F9, F11-F22

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.00	233.43

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
CA SUBSCRIBER PRICE	ENTRY	SESSION
	0.00	-0.59

FILE 'SCISEARCH' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 Institute for Scientific Information (ISI) (R)

FILE 'PASCAL' ENTERED AT 12:50:16 ON 14 MAY 2001
 Any reproduction or dissemination in part or in full,
 by means of any process and on any support whatsoever
 is prohibited without the prior written agreement of INIST-CNRS.
 COPYRIGHT (C) 2001 INIST-CNRS. All rights reserved.

FILE 'BIOTECHNO' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'TOXLIT' ENTERED AT 12:50:16 ON 14 MAY 2001

FILE 'ESBIOBASE' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 Elsevier Science B.V., Amsterdam. All rights reserved.

FILE 'LIFESCI' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 Cambridge Scientific Abstracts (CSA)

FILE 'PROMT' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 Gale Group. All rights reserved.

FILE 'CANCERLIT' ENTERED AT 12:50:16 ON 14 MAY 2001

FILE 'DRUGU' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'TOXLINE' ENTERED AT 12:50:16 ON 14 MAY 2001

FILE 'CABA' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 CAB INTERNATIONAL (CABI)

FILE 'ADISINSIGHT' ENTERED AT 12:50:16 ON 14 MAY 2001
 COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'DRUGUPDATES' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 IMSWORLD Publications Ltd

FILE 'WPIDS' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'WPINDEX' TEMPORARILY UNAVAILABLE

FILE 'ADISALERTS' ENTERED AT 12:50:16 ON 14 MAY 2001
COPYRIGHT (C) 2001 Adis International Ltd. (ADIS)

FILE 'DDFU' TEMPORARILY UNAVAILABLE
If this message appears repeatedly, please notify the Help Desk.
Enter "HELP STN" for information on contacting the nearest STN Help
Desk by telephone or via SEND in the STNMAIL file.

=> d 31-36 ibib abs
NO VALID FORMATS ENTERED FOR FILE 'ADISINSIGHT'
NO VALID FORMATS ENTERED FOR FILE 'DRUGUPDATES'
In a multifile environment, each file must have at least one valid
format requested. Refer to file specific help messages or the
STNGUIDE file for information on formats available in individual
files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d his

(FILE 'HOME' ENTERED AT 11:31:54 ON 14 MAY 2001)

FILE 'REGISTRY' ENTERED AT 11:33:36 ON 14 MAY 2001
L1 1 S MISOPROSTOL/CN

FILE 'STNGUIDE' ENTERED AT 11:34:08 ON 14 MAY 2001

FILE 'HCAPLUS' ENTERED AT 11:35:06 ON 14 MAY 2001
L2 882 S MISOPROSTOL OR L1
L3 145 S (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENI
L4 27636 S SCAN
L5 595 S ((SEX? OR GENITAL) (A) (HYPOACTIV? OR DESIR? OR SATISFACT?
OR
L6 12767 S ((SEX? OR GENITAL) (S) (HYPOACTIV? OR DESIR? OR SATISFACT?
OR
L7 1933 S (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENI
L8 2 S L2 AND L7

FILE 'STNGUIDE' ENTERED AT 11:42:34 ON 14 MAY 2001

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS, USPATFULL' ENTERED AT 11:52:55 ON
14 MAY 2001
E NEAL, GARY/AU
E NEAL GARY/AU
E E18
L9 10 S E18

FILE 'MEDLINE, EMBASE, BIOSIS, USPATFULL' ENTERED AT 11:57:31 ON 14 MAY
2001
L10 43446 S (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR
GENI

L11 6378 S L1 OR MISOPROSTOL
L12 1 S L10 AND L11

FILE 'STNGUIDE' ENTERED AT 12:00:21 ON 14 MAY 2001

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:02:52 ON 14

MAY

2001

FILE 'MEDLINE, EMBASE, BIOSIS, USPATFULL' ENTERED AT 12:03:09 ON 14 MAY 2001

L13 23010 S L1 OR MISOPROSTOL OR (PROTAGLADIN E) OR PGE
L14 55 S L13 AND L10
L15 41 DUP REM L14 (14 DUPLICATES REMOVED)
L16 37 S L13 (S) L10
L17 23 DUP REM L16 (14 DUPLICATES REMOVED)
L18 21 S L17 1999
L19 19 S L17 1998

FILE 'STNGUIDE' ENTERED AT 12:16:42 ON 14 MAY 2001

INDEX 'ADISALERTS, ADISINSIGHT, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, DRUGNL, ...' ENTERED AT 12:20:12 ON 14

MAY

2001

SEA L1 OR MISOPROSTOL OR (PROTAGLADIN E) OR PGE

1241* FILE ADISALERTS
39 FILE ADISINSIGHT
81 FILE AGRICOLA
24 FILE ANABSTR
104* FILE AQUASCI
172 FILE BIOBUSINESS
2* FILE BIOCOMMERCE
9147 FILE BIOSIS
15 FILE BIOTECHABS
15 FILE BIOTECHDS
4490 FILE BIOTECHNO
439* FILE CABA
808 FILE CANCERLIT
5060* FILE CAPLUS
35* FILE CEABA-VTB
1 FILE CEN
76 FILE CIN
361* FILE CONFSCI
0* FILE CROPB
2* FILE CROPU
120* FILE DDFB
2488* FILE DDFU
61* FILE DGENE
120* FILE DRUGB
121 FILE DRUGLAUNCH
125 FILE DRUGMONOG2
19 FILE DRUGNL
2874* FILE DRUGU

14 FILE DRUGUPDATES
 171* FILE EMBAL
 5933 FILE EMBASE
 3570* FILE ESBIODBASE
 0* FILE FOMAD
 0* FILE FOREGE
 11* FILE FROSTI
 14 FILE FSTA
 25* FILE GENBANK
 26* FILE HEALSAFE
 259* FILE IFIPAT
 282 FILE JICST-EPLUS
 3* FILE KOSMET
 3307* FILE LIFESCI
 1* FILE MEDICONF
 4475 FILE MEDLINE
 45 FILE NIOSHTIC
 84* FILE NTIS
 23* FILE OCEAN
 5188* FILE PASCAL
 30 FILE PHAR
 0* FILE PHIC
 476* FILE PHIN
 1371 FILE PROMT
 8868* FILE SCISEARCH
 6 FILE SYNTHLINE
 1693 FILE TOXLINE
 2279 FILE TOXLIT
 3435* FILE USPATFULL
 375 FILE WPIDS
 375 FILE WPINDEX

L20

QUE L1 OR MISOPROSTOL OR (PROSTAGLADIN E) OR PGE

 SEA (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR

GE

 283 FILE ADISALERTS
 69 FILE ADISINSIGHT
 832 FILE AGRICOLA
 2 FILE ANABSTR
 734 FILE AQUASCI
 303 FILE BIOBUSINESS
 2 FILE BIOCOMMERCE
 15038 FILE BIOSIS
 12 FILE BIOTECHABS
 12 FILE BIOTECHDS
 1825 FILE BIOTECHNO
 4136 FILE CABA
 2254 FILE CANCERLIT
 1933 FILE CAPLUS
 2 FILE CEABA-VTB
 11 FILE CEN
 9 FILE CIN
 23 FILE CONFSCI
 117 FILE CROPB
 374 FILE CROPU
 96 FILE DDFB
 421 FILE DDFU
 100 FILE DGENE
 96 FILE DRUGB

8 FILE DRUGNL
 1532 FILE DRUGU
 14 FILE DRUGUPDATES
 152 FILE EMBAL
 13934 FILE EMBASE
 3509 FILE ESBIODBASE
 2 FILE FOMAD
 21 FILE FROSTI
 61 FILE FSTA
 1 FILE GENBANK
 231 FILE HEALSAFE
 57 FILE IFIPAT
 748 FILE JICST-EPLUS
 15 FILE KOSMET
 4182 FILE LIFESCI
 2 FILE MEDICONF
 13531 FILE MEDLINE
 561 FILE NIOSHTIC
 347 FILE NTIS
 185 FILE OCEAN
 4100 FILE PASCAL
 9 FILE PHAR
 1 FILE PHIC
 64 FILE PHIN
 1233 FILE PROMT
 7404 FILE SCISEARCH
 3855 FILE TOXLINE
 3780 FILE TOXLIT
 943 FILE USPATFULL
 91 FILE WPIDS
 91 FILE WPINDEX

L21
 GE

QUE (FEMALE OR WOMAN OR WOMEN OR GIRL OR LADY) (S) ((SEX? OR

 SEA L20 AND L21

1* FILE ADISALERTS
 1 FILE ADISINSIGHT
 0* FILE AQUASCI
 0* FILE BIOCOMMERCE
 19 FILE BIOSIS
 8 FILE BIOTECHNO
 2* FILE CABA
 4 FILE CANCERLIT
 7* FILE CAPLUS
 0* FILE CEABA-VTB
 0* FILE CONFSCI
 0* FILE CROPB
 0* FILE CROPU
 0* FILE DDFB
 1* FILE DDFU
 0* FILE DGENE
 0* FILE DRUGB
 3* FILE DRUGU
 1 FILE DRUGUPDATES
 0* FILE EMBAL
 11 FILE EMBASE
 8* FILE ESBIODBASE
 0* FILE FOMAD
 0* FILE FOREGE

```

0* FILE FROSTI
0* FILE GENBANK
0* FILE HEALSAFE
0* FILE IFIPAT
0* FILE KOSMET
6* FILE LIFESCI
0* FILE MEDICONF
10 FILE MEDLINE
0* FILE NTIS
0* FILE OCEAN
10* FILE PASCAL
0* FILE PHIC
0* FILE PHIN
5 FILE PROMT
18* FILE SCISEARCH
2 FILE TOXLINE
8 FILE TOXLIT
15* FILE USPATFULL
1 FILE WPIDS
1 FILE WPINDEX
L22 QUE L20 AND L21
-----

```

FILE 'SCISEARCH, PASCAL, BIOTECHNO, TOXLIT, ESBIODBASE, LIFESCI, PROMT, CANCERLIT, DRUGU, TOXLINE, CABA, ADISINSIGHT, DRUGUPDATES, WPIDS, ADISALERTS' ENTERED AT 12:27:36 ON 14 MAY 2001

```

L23      605 S F22
L24      79 S L22
L25      69 S L20 (S) L21
L26      222051 S CELLULOSE
L27      0 S L26 (S) L25
L28      0 S L26 AND L25
L29      36 S L25

```

FILE 'STNGUIDE' ENTERED AT 12:49:45 ON 14 MAY 2001

FILE 'SCISEARCH, PASCAL, BIOTECHNO, TOXLIT, ESBIODBASE, LIFESCI, PROMT, CANCERLIT, DRUGU, TOXLINE, CABA, ADISINSIGHT, DRUGUPDATES, WPIDS, ADISALERTS' ENTERED AT 12:50:16 ON 14 MAY 2001

=> d 129 31-36 ibib abs

NO VALID FORMATS ENTERED FOR FILE 'ADISINSIGHT'

NO VALID FORMATS ENTERED FOR FILE 'DRUGUPDATES'

In a multifile environment, each file must have at least one valid format requested. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.

REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):

REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):end

=> d 129 31-36

```

L29  ANSWER 31 OF 36  DRUGU  COPYRIGHT 2001 DERWENT INFORMATION LTD
AN   1998-10539  DRUGU   T E
TI   Antigestagens: Possible gynecological and obstetric uses.
AU   Ortman O; Emons G; Schulz K D
CS   Univ.Philipps-Marburg; Univ.Lubeck
LO   Lubeck; Marburg, Ger.
SO   Med.Welt (48, No. 12, 525-29, 1997) 1 Fig. 39 Ref.
      CODEN: MEWEAC      ISSN: 0025-8512

```

AV Medizinische Universitaet zu Luebeck, Ratzeburgerallee 160, D-23562
Luebeck, Germany.
LA German
DT Journal
FA AB; LA; CT
FS Literature

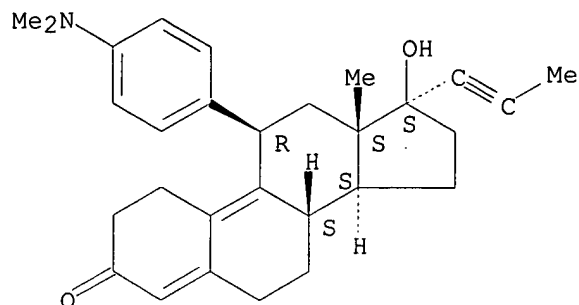
L29 ANSWER 32 OF 36 DRUGU COPYRIGHT 2001 DERWENT INFORMATION LTD
AN 1992-10958 DRUGU T S E
TI The Management of Non-Steroidal Anti-Inflammatory Drug-Induced
Gastroduodenal Ulcers.
AU Berge Henegouwen G P van; Smout A J P M
LO Utrecht, Netherlands
SO Scand.J.Gastroenterol. (26, Suppl. 188, 87-91, 1991) 4 Tab. 30 Ref.
CODEN: SJGRA4 ISSN: 0036-5521
AV P.O. Box 85500, 3508, GA Utrecht, The Netherlands.
LA English
DT Journal
FA AB; LA; CT
FS Literature

L29 ANSWER 33 OF 36 CABA COPYRIGHT 2001 CABI
AN 81:1574 CABA
DN 800154773
TI Lordosis induction in the rat by prostaglandin E2 systematically or
intracranially in the absence of ovarian hormones
AU Rodriguez-Sierra, J. F.; Komisaruk, B. R.
CS Institute of Animal Behavior, Rutgers State University, 101 Warren
Street,
Newark, New Jersey 07102, USA.
SO Prostaglandins, (1978) Vol. 15, No. 3, pp. 513-524. 24 ref.
ISSN: 0090-6980
DT Journal
LA English

L29 ANSWER 34 OF 36 CABA COPYRIGHT 2001 CABI
AN 78:3305 CABA
DN 780135525
TI Facilitation of lordosis in the rat by prostaglandin E2
AU Dudley, C. A.; Moss, R. L.
CS Department of Physiology, Texas University Health Science Center,
Southwestern Medical School, Dallas, Texas 75235, USA.
SO Journal of Endocrinology, (1976) Vol. 71, No. 3, pp. 457-458. 8 ref.
ISSN: 0022-0795
DT Journal
LA English

L29 ANSWER 35 OF 36 ADISINSIGHT COPYRIGHT 2001 (ADIS)
ACCESSION NUMBER: 1998:1453 ADISINSIGHT
SOURCE: Adis R&D Insight
DOCUMENT NO: 001657
CHANGE DATE: Jan 31, 2001
GENERIC NAME: Mifepristone
SYNONYM: Mifeprex; RU 38486; RU 486
CHEMICAL NAME: Estra-4,9-dien-3-one, (11beta,17beta)-11-(4-
(dimethylamino)phenyl)-17-hydroxy-17-(1-propynyl)-
TRADE NAME: Mifegyne
MOLECULAR FORMULA: C29 H35 N O2
CAS REGISTRY NO.: 84371-65-3
STRUCTURE:

Absolute stereochemistry.



EPHMRA ATC CODE: G2 Other gynaecologicals; L2B Cytostatic Hormone Antagonists
WHO ATC CODE: G03D Progestogens; L02B Hormone Antagonists and Related Agents
HIGHEST DEV. PHASE: Launched

COMPANY INFORMATION
ORIGINATOR: Aventis Pharma (France)
PARENT: Aventis
LICENSEE: Cosan; Exelgyn; Nonindustrial source; Unknown
OTHER: Contragest; Shanghai Hualian Pharmaceutical

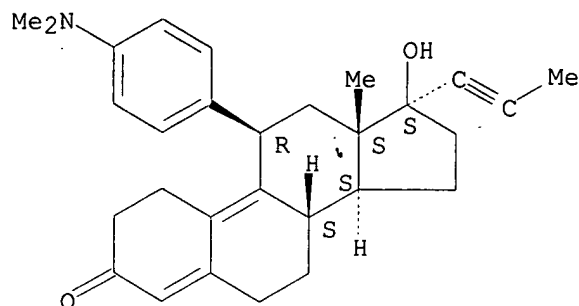
OTHER SOURCES: 800671416; 800802900; 800161315; 800238704; 800538779; 800833061; 800690334; 800723592; 800745348; 800813601; 800821978; 800698668

WORD COUNT: 3041

L29 ANSWER 36 OF 36 DRUGUPDATES COPYRIGHT 2001 IMSWORLD

ACCESSION NUMBER: 93:1064 DRUGUPDATES
SOURCE: R&D Focus, (27 Nov 2000)
GENERIC NAME: mifepristone
REFERENCE: INN
LABORATORY NAME: RU 486; RU 4866; RU 38486; R 38486
TRADE NAME: MIFEGYNE; MIFEPREX
CHEMICAL NAME: (11beta,17beta)-11-[4-(dimethylamino)phenyl]-17-hydroxy-17-(1-propynyl)estra-4,9-dien-3-one
CAS REGISTRY NO.: 84371-65-3
STRUCTURE:

Absolute stereochemistry.



DERIVATIVE(S): 84371-65-3 mifepristone
 91934-98-4 N-oxide
 105868-38-0 cpd with butyl acetate (1:1)
 108179-34-6 fumarate (1:1)
 127888-97-5 mixt cont
 127888-96-4 mixt with EPA
 127888-95-3 mixt with (Z,Z,Z)-8,11,14-eicosatrienoic acid
 93781-44-3 (11beta,13alfa,17beta)
 91935-24-9 (3E,11beta,17beta), oxime
 91935-23-8 (3Z,11beta,17beta), oxime
 96346-56-4 (11beta,13alfa,17alfa)
 116948-77-7 (11beta,14beta,17alfa)
 101978-06-7 (8alfa,11alfa,13alfa,14beta,17alfa)
 122742-25-0 replaced by 84371-65-5
 83203-42-3 replaced by 84371-65-3

CLASSIFICATION: G2X9 Other Gynecologicals; L2B9 Other Cytostatic Hormone
 Antagonists; G3A Hormonal Contraceptives, Systemic
 HIGHEST DEV. PHASE: Marketed (80)

COMPANY INFORMATION:

Type	Company	Nationality	Region
Originator	Aventis	France	
Licensee	Exelgyn	France	Worldwide outside the USA
Other	Cosan	Switzerland	Switzerland
Other	Danco	United States	United States
Other	Population Council	United States	United States
Assignee	Roussel Uclaf		

=> log y

COST IN U.S. DOLLARS

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

CA SUBSCRIBER PRICE

SINCE FILE	TOTAL
ENTRY	SESSION
49.19	282.62

SINCE FILE	TOTAL
ENTRY	SESSION
0.00	-0.59

STN INTERNATIONAL LOGOFF AT 12:53:23 ON 14 MAY 2001

Connection closed by remote host